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THE TREATMENT OF TULAREMIA WITH STREPTOMYCIN—REPORT OF THREE CASES

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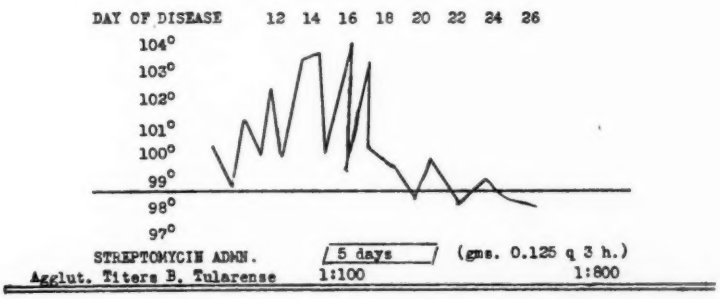
During the past year scattered reports have appeared in the literature regarding the treatment of all forms of tularemia with streptomycin. Up to the present time, there is every indication that this new antibiotic will prove just as effective in the control of this infection as preliminary laboratory studies indicated.

Foshay and Pasternack[1] reported seven cases of streptomycin treated tularemia in February, 1946, and emphasized the fact that this drug definitely altered the natural course of the infection. According to these authors, without specific treatment, "the disease usually causes thirty-one days of fever, thirty-one days in bed, a duration of disease of four months, characterized by buboes for 3.5 months, disability for 3.5 months, and healing time for primary lesions of thirty-nine days." Cecil's Textbook of Medicine, Second Edition, published in 1931, notes that the convalescence is slow, and states that it is rare for a patient to be at work again for the end of a month, while some have not returned to normal for six months or even a year.

All but one of the cases reported here were well in less than a month after the onset, and the third case, though symptom-free at the end of the third week, had a small draining sinus from an infected bubo on the back of the neck, which was healed completely at the end of two months.

CASE NO. I



The streptomycin used in the first two cases was furnished through the National Research Council; the last case was seen after the drug was released for general use. Cases one and three received a total of five grams, given in divided doses of 0.125 gms. each, intramuscularly, every three hours. Case two was treated for seven days, getting a total of seven grams. No toxic reactions were seen.

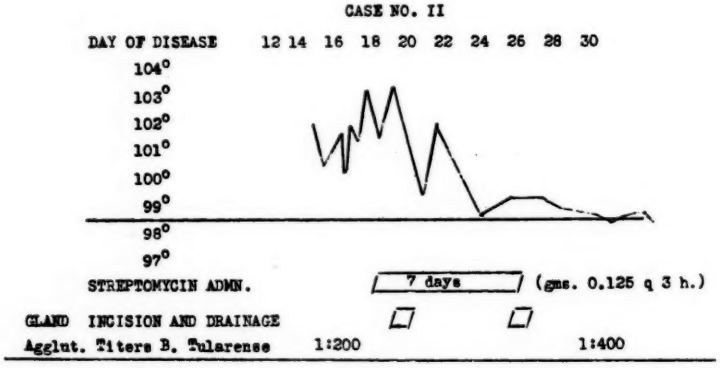
CASE I: J. C. T. was a healthy, twenty-eight year old male, who received a tick bite under the right

arm on May 2, 1946. Three days later there was pain and redness around the site of the bite and he had a chill, followed by a high fever. For the next eleven days he had chills and fever every other day, but was feeling a little better on May 16, 1946, when he was admitted to the hospital. He complained of some pain in the head, and generalized aching, and noticed that with his chills and elevation of temperature, the sore under his arm always became more painful and red. The admission oral temperature was 100.2° and the pulse 86 per minute. There was an ulcerated skin lesion in the posterior axilla on the right, without any noticeable regional lymph node involvement. The skin surrounding the lesion was edematous and dark red. The remainder of the physical examination was negative. The urine was normal, the hemoglobin 98 per cent and the white blood count 9,600, with a normal differential. The Kahn precipitation test was negative.

From the day after admission, twelfth day of the disease, the temperature rose steadily to a peak of 103.8° on the sixteenth day, when streptomycin was started. Two days later the temperature came down promptly and the patient began to feel better. The skin lesion looked dry and the ulcer began to heal from the bottom. At the conclusion of the treatment, which covered a period of five days, the patient felt well and remained out of bed most of the day. Agglutinations for B. tularens were positive 1:100 on the sixteenth day of the disease and on the twenty-second day the titer was 1:800.

Two weeks from the time of admission, the patient left the hospital. When seen on June 28, 1946, and again on December 29, 1946, he was entirely well.

CASE II: S. R., a white male, aged 8 years, lived in the country, kept tame rabbits and frequently found ticks in his hair. He was a neighbor of CASE I, and they were partners in the rabbit business. On May 12, 1946, his mother noticed a small, infected bump in the hair on the back of his head and picked it with a needle. That evening the child's temperature was elevated and the infected area was spreading. He continued to have a high fever for three days and then felt better for thirty-six hours. After that the temperature rose to over 103 degrees and remained high until the end of the second week of his illness (May 26, 1946), when he was admitted to the hospital. At that time a large swollen lymph gland had appeared just below the infected area on the back of the head. The temperature was 101 degrees on admission, and the child was fretful and nervous. There was a large, encrusted lesion on the upper right posterior surface of the neck. The regional cervical lymph nodes were enlarged and tender. The spleen could not be felt. Blood taken on the fifteenth day of the disease, May 27, 1946, gave a positive agglutination test for B. tularens 1:200.



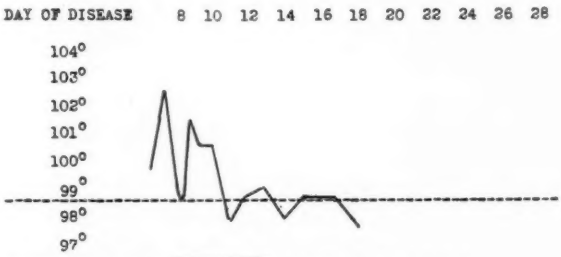
Streptomycin was started on the twenty-ninth day of May, the eighteenth day of the disease, but the temperature rose from 101° to 103°. At first it was thought that the increased temperature was due to drug sensitivity, but later it was found that one of the infected buboes was tender and fluctuating. On June 2, 1946, the gland was incised and drained large quantities of thick, creamy pus. On the following day, the temperature was normal and the patient felt well except for some soreness in the neck. After that his temperature was never over

99.2° and he remained well. The lesion on his neck healed but there was a small draining sinus which persisted for one month after release from the hospital.

CASE III. H. T. R., a 59 year old farmer, had been well until December 5, 1946. On November 23, 1946, he had cut his left thumb. This had been slow healing and cracked open frequently when he used his hands. On December 4, 1946—eight days before admission to the hospital—he found his dogs scuffling over a dead rabbit, so he divided the carcass between them. In doing this, he smeared quite a lot of blood on both of his hands. One day later he began to have a hard chill which was followed by a high fever. The day after that he noticed that there was considerable swelling of the left thumb and the gland under his left arm was swollen and tender. He continued to have some elevation of temperature and generalized discomfort until December 12, 1946, when he was admitted to the hospital. His temperature, which was 99.8° on admission, soon rose to 102.4°. There was some swelling at the base of the left thumb and pus could be seen under the skin edge. The axillary node was red, hot and tender. Total white blood count was 12,500, with a fairly normal differential count. The gross examination of the urine was negative, but the microscopic showed 6-10 WBC and 5-6 RBC per high power field. Agglutinations were negative on the thirteenth day of the disease (December 18, 1946). Continuous hot wet dressings were started, and because his history strongly suggested the possibility of tularemia, he was given streptomycin intramuscularly, 0.125 gms, every three hours. Two days later his temperature was normal, and he was sitting up in a chair. He left the hospital on December 18, 1946, feeling well. The lesion on the thumb had healed and the enlarged axillary node had almost disappeared.

A report from blood sent in for agglutinations by the family physician on December 30, 1946, showed a positive test for B. tularens 1:200.

CASE NO. III



STREPTOMYCIN ADMN. / 5 days / (gms. 0.125 q 3 h.)

Agglut. Titers B. Tularense Negative (12-18-46) 1:200 (12-31-46)

SUMMARY AND CONCLUSIONS

Three cases of tularemia were treated with streptomycin and recovered satisfactorily.

The first two cases were probably contracted from an infected tick; the third from an infected rabbit.

CASE II responded to streptomycin only after an infected regional lymph gland had been incised and drained.

CASE III was interesting because of the unusually short period of incubation, symptoms appearing 24 hours following contact with an infected rabbit.

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Professional Building.

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SOME OBSERVATIONS ON THE CLASSIFICATION AND DIAGNOSTIC CRITERIA OF HEART DISEASES*

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INTRODUCTION

Early classifications and criteria for cardiac diagnosis published by White and Myers in 1921[1] and Wycoff and Lingg in 1926[2] form the groundwork for our present nomenclature and criteria for cardiac diagnosis as published by the American Heart Association[3]. Although there still remain a number of gaps in our knowledge of heart disease etiologically, the possibilities of a rather precise cardiac diagnosis in the majority of patients with heart disease have increased tremendously during the past two decades.

In order to be complete, a cardiac diagnosis should include first the *etiological* basis for the disease; second, the *anatomical* lesion or lesions present; and, third, the disturbance in cardiac function that is present. We may go even further and classify patients according to the functional capacity of the heart and/or the degree of restriction of physical activity required.

It is not sufficient to simply make a diagnosis of rheumatic heart disease without some further clarification of the structural damage that has occurred to the heart or heart valves; nor should we make an isolated diagnosis of auricular fibrillation or congestive heart failure without indicating the etiologic and anatomic bases for these disturbances in function if known. Alone, they are incomplete as cardiac diagnoses and constitute only disturbances in the cardiac mechanism or function.

It would be impossible within the scope of this paper to cover the entire field of cardiovascular diagnosis, so my purpose will be to call attention to some of the more commonly encountered etiological types of heart disease and to offer some practical suggestions which may be helpful in differentiating them one from another.

HYPERTENSIVE HEART DISEASE

A male patient, aged 49, presented himself for diagnosis, complaining of shortness of breath on effort of increasing severity over a period of four months, and with a past medical history that afforded no helpful clues except fairly frequent headaches and the fact that he was turned down for life insurance at age 45. From this point our search begins with two symptoms—dyspnea and headache. In many instances dyspnea heralds the onset of cardiac insufficiency, but we must remember that dyspnea may be due to other causes than heart disease (such as pulmonary disease or severe anemia) and that the subjective sensation of "shortness of breath" as described by a patient may, in fact, be the feeling of anxiety and inability to get a deep breath, often experienced by neurotic individuals or those with neurocirculatory asthenia. It is important to establish the difference between true dyspnea and the sighing type of respirations encountered in neurotics and those with neurocirculatory asthenia. This particular patient had true dyspnea for which no extracardiac cause was apparent. So we have a symptom that is presumably the result of a disturbance in cardiac function. We must now determine by physical examination and other diagnostic procedures what anatomical changes, if any, have taken place in the cardiovascular system. Physical examination revealed moderate tortuosity of the retinal vessels with some arteriovenous nicking; the radial arteries felt somewhat hard and incompressible to palpation; the blood pressure measured 210 systolic and 120 diastolic; the cardiac apex impulse was forceful and the point of maximum impulse was in the 5th interspace 1½cm. outside the midclavicular line, indicating definite enlargement. The urine showed a 2+ reaction for albumin and a few hyaline and granular casts in the sediment. A 6 ft. film of the chest for heart size and contour confirmed the physical finding of cardiac enlargement and revealed some tortuosity of the aorta. The electrocardiogram showed well-marked left axis deviation with some inversion of the T-waves in lead I. a definite hypertensive pattern.

Altogether these findings are typical of full blown hypertensive heart disease, and we need search no further to establish a complete cardiovascular diagnosis which was recorded as follows:

*Read before the annual meeting of the Old Dominion Medical Society, at Roanoke, Va.

1. Hypertensive heart disease to cover the *etiologic* diagnosis.

2. Moderate cardiac enlargement and tortuosity of the aorta to indicate the *anatomic* changes that have occurred, and we might add, parenthetically, the fact that vascular damage was present in the eye grounds (hypertensive retinopathy) and kidneys (nephrosclerosis) and,

3. Finally, left ventricular weakness, or failure, to indicate the *functional* status of the heart.

To arrive at such a diagnosis is comparatively simple when so many changes have occurred to point to the correct etiologic, anatomic, and functional bases for the disease. However, it is not always easy to assess the extent of damage that has occurred to the cardiovascular-renal system from hypertension when the heart and kidneys are still fully compensated and the patient essentially symptom free. It is during this stage of the disease that we must furnish our patients with a careful hygienic program and be constantly on the alert for danger signals that might indicate further curtailment of their activities or symptomatic or supportive measures.

CORONARY HEART DISEASE

A second patient, a male, aged 54, gave a history of pain beneath the breast bone occurring on effort, particularly after meals, and relieved by cessation of activity, which symptom had been present for six or seven months. Several hours before I saw him, while sitting at his desk, he developed a "heavy, squeezing" sensation beneath the lower end of the sternum which gradually increased in severity, spread across the chest, and radiated down the left arm. The pain persisted, was attended by cold perspiration, pallor, restlessness, and inability to obtain relief from repeated doses of soda. Later he became nauseated and vomited several times. Only after the administration of atrophine, gr. 1/100, aminophyllin, gr. 7½, and 3/4 gr. of morphine over a period of one hour did he obtain any relief from his distress. His blood pressure measured 90/60.

With such a clinical history, none of us would fail to recognize the classical picture of coronary thrombosis or occlusion, but, if we reflect for a moment, it is apparent that coronary thrombosis or occlusion signifies only the *anatomic* lesion responsible for such a disaster. So if we are consistent, we must once more indicate the etiologic basis for the changes present, which in the vast majority of cases is arteriosclerosis. The complete cardiac diagnosis in this particular instance included the following:

1. *Arteriosclerotic or coronary* heart disease (etiologic)

2. Coronary occlusion with myocardial infarction (anatomic)

3. Anginal syndrome (indicating a *functional* insufficiency of the heart due to narrowing and/or spasm of the coronary arteries).

Volumes have been written concerning the differential diagnosis of chest pain, but we can never overemphasize the importance of this problem that constantly confronts us in practice. To mention only a few of the conditions that may be confused with coronary insufficiency or occlusion, they include:

1. Pulmonary embolism, an ever present complication in surgical patients, and more frequently present in medical patients than was formerly thought

2. Aortitis

3. Dissecting aneurysm of the aorta

4. Diaphragmatic hernia

5. Pericarditis

6. Cardiospasm

7. Muscle pains in the chest

8. The "heart-ache" that may be present in neurotic individuals as well as in those with large hearts from organic disease

9. And other conditions which may even be below the diaphram, such as acute gall bladder disease.

Time does not permit a detailed discussion of each of these conditions, but the points I want to make are that the pain of angina pectoris is (1) typically beneath the sternum, with or without radiation to one or both arms; (2) is almost invariably related to exercise, excitement, or meals (particularly heavy meals); and, (3) subsides with cessation of activity or following the use of nitroglycerin beneath the tongue. Any substernal pain which persists for thirty minutes or longer and is attended by signs of shock had better be regarded as being due to coronary occlusion until proved otherwise. Clinically, the pain of coronary occlusion usually begins with mild intensity, gradually building up to great severity over a period of several minutes or longer, which may sometimes distinguish it from the pain of pulmonary embolism or dissecting aortic aneurysm in which the pain strikes, as has been described by one patient, "like the blow of a sledge hammer" with immediate agonizing suddenness. Although well-

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marked coronary thrombosis is more likely than not to be attended by severe pain, there are exceptions —without any pain at all; in such cases there may be dyspnea instead or simply collapse or prostration. One further highly significant point in the diagnosis of angina pectoris or coronary occlusion concerns the sex of the patient. It is a clinical aphorism that "female patients under the age of 50 *rarely* have coronary disease in the absence of hypertension." Therefore, in any woman under the age of 50 whose presenting complaint is substernal pain, one can be reasonably sure it is *not* angina pectoris or due to coronary disease if the blood pressure is normal.

PULMONARY HEART DISEASE

Not long ago I saw a man, aged 54, who had been laid up for several weeks because of thrombophlebitis of some greatly dilated varicosities of the right leg. His local physician had treated him with penicillin intramuscularly and hot applications locally with good results, and most of the pain, tenderness, and local heat had subsided in his leg. Then one evening while his doctor was present, he flexed and then extended his leg to demonstrate that it felt better. Almost immediately he was taken with rather severe substernal pain attended by shortness of breath, sweating, pallor, rapid pulse and later some pain in the left lower chest. The substernal pain subsided in a few minutes, and his color improved, but the rapid pulse continued. The electrocardiogram showed no remarkable abnormality, but, because of the brief time that had elapsed since the onset of the pain, this finding could not be relied upon to rule out coronary occlusion. It seemed more likely that a small part of the clot had been dislodged from the thrombophlebotic area following the flexion and extension of his leg and had lodged in his pulmonary circulation. He was immediately hospitalized and the long saphenous vein tied off without further developments. A chest film on the day after admission showed evidence of pulmonary infarction in the left lower lung field. Experience has shown that we might well have gone a step further and tied off the superficial femoral vein to take care of any possible extension of the process from the superficial to the deep veins.

This case emphasizes the potential danger of peripheral vascular disease as a source of pulmonary vascular accidents which may prove fatal when emboli are of sufficient size to obstruct one of the major pulmonary vessels. Fortunately for this patient, the embolus was small, but there was no assurance that larger, more serious ones might not have broken loose unless vein ligation was accomplished. When larger elements of the pulmonary vascular tree are the site of embolism, the sudden burden that is placed on the right ventricle may cause it to dilate acutely and fail. This condition, the classical description of which was made by McGinn and White[4] some ten years ago, is called *acute cor pulmonale* and is characterized by definite physical and electrocardiographic findings. Theoretically, some degree of acute cor pulmonale exists with any pulmonary embolus, but the severity of symptoms and the gravity of the prognosis are in direct proportion to the size of the embolus, other things being equal. Many sudden, tragic deaths from pulmonary embolism are now preventable, and we must be alert to this possibility in both surgical and medical patients who experience sudden, repeated bouts of unexplained chest pain, particularly when thrombophlebitis or phlebothrombosis is known to be present. Moreover, we should be mindful of pulmonary embolism at all times in the differential diagnosis of chest pain even in the absence of signs of peripheral vascular disease.

The management of thrombophlebitis and phlebothrombosis is the subject of some controversy, the pros and cons of which would carry me too far afield. There are strong advocates of wholesale vein ligation; others prefer lumbar sympathectomy, and yet another group use the anticoagulant preparations, such as dicumarol and heparin. Each have their good points and bad and their particular indications. When controversies exist in such matters, it is perhaps wisest to be conservative when possible, use the method in which you are most skilled, and await developments in the field.

RHEUMATIC HEART DISEASE

A young woman in her early 30's consulted me recently complaining of severe palpitation and increasing dyspnea on effort. She was married and had three young children, and the pressure of her responsibilities at home was great. She tired easily and could not sleep at night because of distressing palpitation. She had never been told that there was anything wrong with her heart during any of her pregnancies nor given any advice concerning the wisdom of further childbearing. She denied having had rheumatic infection but did give a his-

tory of "growing pains" during childhood. Physical examination revealed a thin, but fairly well-developed young woman who did not appear acutely ill. Her heart was definitely enlarged downward and to the left with a forceful apex impulse that was visible and palpable. Along the left sternal border, maximal in the 4th interspace, there could be heard an early, high-pitched diastolic murmer of grade III to IV intensity. The pulse rate was 85, the rhythm dominantly regular but interrupted by frequent premature beats, and the pulse at the wrist was Corrigan in character. The blood pressure measured 140/60, a rather high pulse pressure. Physical findings were otherwise not remarkable. The serology of the blood was negative.

In the absence of syphilis, these findings are altogether typical of rheumatic heart disease with aortic regurgitation, well-marked cardiac enlargement, a definite impairment of myocardial reserve and frequent premature beats which by electrocardiogram proved to be ventricular in origin.

It seems almost incredible that a person with such definite heart disease could go through three pregnancies with hospitalization for delivery each time and have it go undiscovered—yet such things do happen, sometimes with tragic endings. Unless this young woman's circumstances permit her to lead a very careful life, she will be dead within five years or less, and we will be to blame for not having discovered a potentially fatal heart lesion before allowing her to add the burden of three pregnancies to an already damaged heart.

The two most important and significant heart murmurs that occur in rheumatic heart disease are (1) that of aortic regurgitation, present in the case just discussed, and (2) the murmur of mitral stenosis. Both are diastolic in time, but the murmur of aortic regurgitation is early in diastole, high-pitched in character, best heard with the diaphragm type of stethoscope, along the left sternal border, while the murmur of mitral stenosis is late in diastole, low-pitched or rumbling in character, and is best heard with the bell stethoscope at the apex region of the heart. Both murmurs are very characteristic and, once you are sure of their nature, they can be recognized as easily as the bark of a dog. It is impossible to go further in helping one to recognize these murmurs. The only way to develop skill and confidence is to listen to them whenever the opportunity presents itself.

OTHER ETIOLOGIC TYPES OF HEART DISEASE

The foregoing etiological types of heart disease are commonly encountered in practice and, along with luetic heart disease, make up the majority of cases.

Congenital heart disease is less commonly encountered, but is worthy of mention, not only because of the great advances that have been made in the earlier recognition of the various developmental abnormalities, but even more so because of the brilliant work of Gross[5], Blalock[6] and others in the surgical treatment of patent ductus arteriosus, coarctation of the aorta, and a variety of other congenital abnormalities in which pulmonary stenosis is the dominant lesion. This work is outstanding and now affords additional means of reversing the ravages of heart disease which all too often we are unable to do.

It is rare now to encounter a patient whose cardiovascular disease is due to thyrotoxicity because of the general alertness of the profession to the manifestations of hyperthyroidism and the relatively early subtotal thyroidectomy which forestalls the cardiovascular complications of the disease.

Likewise, because of the therapeutic means at our disposal, it is unusual any longer to encounter the myxedema heart. Other less common conditions which may implicate the cardiovascular system include anemia, bacterial infection, neoplasm, thoracic deformities, toxic agents and trauma.

CARDIAC NEUROSIS (PSYCHONEUROSIS)

In recent years, with the increasing incidence of heart disease and the widespread publicity about it, the problem of cardiac neurosis has assumed a place of great importance in medical practice. Almost daily we are confronted with cases that must be carefully studied and treated with this condition in mind, and, not infrequently, anxious members of a family may be as difficult to manage as the patient himself.

Cardiac neurosis (or psychoneurosis) in which there is actual fear or anxiety about the heart should not be confused with the equally common condition known as neurocirculatory asthenia, although quite often the two occur together and eventually the latter may cause concern about the heart in the patient, his family or his physician. Any neurotic or psychoneurotic state may have cardiovascular symptoms associated with it or be in part or completely based on the belief of the presence of heart disease even

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with no symptoms whatever. In neurocirculatory asthenia, such symptoms as palpitation, precordial pain, dyspnea of the sighing type, faintness, dizziness, tremor, sweating and nervousness may be present to a marked degree without any particular apprehension about the heart. However, almost without exception, an anxiety neurosis of some sort is present in such patients and, without proper guidance and psychotherapy, a cardiac neurosis may develop in the course of time.

Underlying every cardiac neurosis there is, as a rule, some definite exciting factor, The occurrence of heart disease, especially sudden heart death among relatives or friends or even simply reported in the newspapers under dramatic headlines, may initiate a cardiac neurosis in one who is ready for a neurosis of some sort. The finding by a physician of a heart murmur, trivial or not, of some disturbance of rhythm which may be insignificant, of hypertension great or slight, or of actual heart disease, may be the exciting spark. Undue emphasis on some detail or actual misinterpretation of X-rays or electrocardiograms may provide the stimulus in the patient's mind. Subjective sensations may be the starting point, such as a disagreeable premature beat, a paroxysm of tachycardia, or the numerous symptoms of neurocirculatory asthenia. Likewise, various pains in the chest of cardiac or non-cardiac origin may create fear of heart disease and, therefore, in the minds of most laymen fear of sudden death. It is important to recognize and treat these cases early, for a long established cardiac neurosis, particularly when complicating serious heart disease, may be almost incurable.

Some years ago I saw a young man, 32 years old, who was overweight and had been overworking and smoking heavily. His father had angina pectoris. About four months before I saw him he first began to be bothered by a little dyspnea in the form of air hunger with s i g h i n g respiration, occurring at most any time but mostly when very tired from overwork. One month later while in a barber's chair having a haircut, he was seized by a sense of suffocation and after the haircut he had to rush outdoors for air. He then consulted a doctor who found him breathing deeply and rapidly. He was put to bed where he remained for four weeks with much sighing respiration at first and then gradual recovery. He was then seen by a local specialist who made a diagnosis of "soldier's heart" after obtaining a normal electrocardiogram. But because of the persistence of some sighing respiration, he went to a distant clinic for study. An electrocardiogram taken there was said not to be wholly normal, and he returned home with the impression that he was a probable "heart case." Following that he was in a state of nervous apprehension, the sighing continued, and a new symptom consisting of a little heartache was present for four weeks.

Except for apparent nervousness and obesity, physical examination was quite normal. The heart was normal in size, shape and sounds. There were no murmurs. The pulse was regular at a rate of 84. The blood pressure measured 140 systolic and 85 diastolic. A subsequent electrocardiogram was normal.

Here we have an example of a case of marked cardiac neurosis set off by a fear both by himself and others that his sighing meant heart disease. Unfortunately, while he was convalescing from this state of mind and body, his apprehension was stimulated again by overemphasis of some trivial variation of his electrocardiogram from the usual average normal. There was no evidence of real heart disease.

This sort of thing is going on about us all the time. The circumstances may vary but essentially the mechanism is the same, namely, fear and anxiety about cardiac invalidism and sudden death. These people need careful attention, tempered by judicious sympathy and an optimistic attitude toward the future, and much can be accomplished in giving hope, encouragement and a new lease on life to persons who might otherwise be almost totally incapacitated by ailments which have no organic basis. And, in accomplishing this mission, we cannot be very reassuring to a patient who is worried and apprehensive about his heart if he is asked to return for office visits too frequently. Of course, some patients need a great deal of reassurance, but it has never seemed very consistent to me to tell a patient that he has nothing to worry about and then have him return for weekly office visits. If careful examination gives reasonable assurance that no significant heart trouble is present, one can be much more reassuring by asking the patient to return in six months or a year. Then, if you have acquired his confidence and have been convincing in your statements to him, he cannot help but feel optimistic toward the future.

SUMMARY

By means of case presentations the value of the everyday use of some sort of classification in cardiac diagnosis has been emphasized. Although some differences of opinion naturally exist concerning the details, the present edition of the "Nomenclature and Criteria for Diagnosis of Diseases of the Heart"[3] published by the American Heart Association is widely used and is as inclusive as one might need for practical purposes.

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820 S. Jefferson Street.

Pneumonia Following Chest Injury Preventable.

Pneumonia, following injury to the chest, can be prevented with large doses of penicillin or sulfadiazine, according to Edward Phillips, M.D., of Oakland, Calif.

Writing in the January 18 issue of *The Journal of the American Medical Association,* Dr. Phillips presents his observations on 73 patients treated at Permanente Foundation Hospital for pneumonia following injury to the chest.

Of these 73 patients, 43 had fractured ribs, one had a fractured shoulder blade and one had a fractured breast bone.

The author cites two investigators who demonstrated by animal experimentation how a blow to the chest, with or without fracture of the ribs, can produce injury to the lungs. The blow stimulates the vagus nerve, which extends from the cranium to the lungs, to contract the bronchial tubes which results in a partial collapse of the lung. This collapse area becomes infected because it lacks aeration.

Injections of atropine into the veins, which paralyzes the terminal fibers of the vagus nerve, will prevent or minimize the chances of collapse and infection following injury to the chest, states the author. Large doses of penicillin and sulfadiazine will sterilize the lung area.

Additional facts regarding the 73 patients follow:

—In 54 patients (74 per cent) pneumonia developed only on the same side as the injury.

—The majority of patients had a mild type of pneumonia.

—Over 92 per cent of pneumonia cases occurred within six days of the injury.

—Over 50 per cent of the patients recovered within one week.

—Three patients died with a resultant mortality rate of 4.1 per cent.

Twenty-six consecutive patients with severe chest and lung injuries who were treated with large doses of either penicillin or sulfadiazine did not contract pneumonia or other complicating infections.

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RUTIN IN THE TREATMENT OF INCREASED CAPILLARY FRAGILITY: A PRELIMINARY REPORT*

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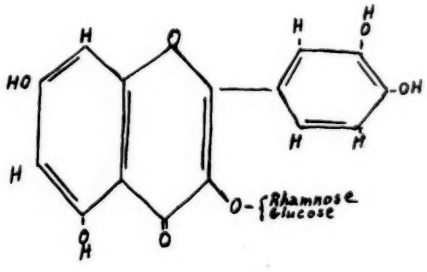
In 1936, and again in 1938 and 1940, Paterson,[1] Wartman,[2] Winternitz *et al.*,[3] reported the association of capillary rupture with intimal hemorrhage in the causation of cerebral and coronary vascular lesions. In 1940, Paterson[4] stated: "Capillary rupture with intimal hemorrhage is intimately concerned with the mechanism of cerebral arterial thrombosis and possibly, in certain cases, with the causation of cerebral arteriospasm and rupture. It is suggested that the factors responsible for the rupture of intimal capillaries in the cerebral arteries are high intracapillary pressure from hypertension, progressive atheromatous degeneration of the supporting tissue and increased capillary fragility from a variety of causes." Griffith and Lindauer[5,8], therefore, thought it possible that certain of the vascular accidents which sometime occur in cases of hypertension might be produced by an abnormal condition of the capillaries and in those patients with increased capillary fragility it was noted that crude hesperidin, a flavone glucoside, given orally restored the fragility to normal. Prior to this, Szent-Gyorgyi and others[6] reported that Citrin, or "Vitamin P" (a mixture of flavone derivatives including among other substances hesperidin, eriodictin and quercitrin plus ascorbic acid), had a tendency to regulate capillary permeability.

MATERIAL

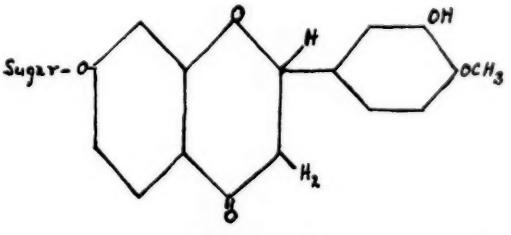
Rutin is a flavonol glucoside and has been reported to be a constituent of thirty-three species of plants, including buckwheat, forsythia, tobacco, garden rue, violets and elder flowers. Suspecting that the active constituent of crude hesperidin is of a flavonol nature, Griffith, Couch, and Lindauer first applied rutin clinically in 1942. The drug has been studied chemically by Couch and his co-workers.[7] Neither acute nor chronic toxicity to laboratory animals have been observed. The chemical formula of rutin is $C_{27}H_{30}O_{16}3H_2O$ and structurally, it is related to hesperidin (Fig. 1).

Rutin is a tasteless yellow powder consisting of needle-shaped crystals melting at 185° to 192° C. and decomposing at 214-215°. It is quite stable but gradually darkens on exposure to light. It is slightly soluble in cold water, more soluble in boiling water, alcohol, and acetone, and very soluble in alkaline solution. It is insoluble in ether, benzene, and chloroform. On hydrolysis with dilute acids it yields quercetrin, glucose, and rhamnose.

Fig. 1



RUTIN
(C_{27} H_{30} O_{16})



HESPERIDIN

The rutin used in this study was prepared by the Eastern Regional Research Laboratory of the U. S. Department of Agriculture. Flue-cured tobacco originally, and later fresh buckwheat plants, were used as the sources of rutin. It was extracted by alcohol percolation. Rutin was preferred to crude hesperidin since it is a pure substance amenable to accurate dosage and can be given in smaller doses, 20 mg. as against 500 mg. for the crude hesperidin.

METHOD

Capillary fragility as evidenced by the Petechial Index was determined by Griffith and Lindauer's modification of Göthlin's method.[5]

*Read before the Scientific Meeting, McGuire Clinic, Richmond, Virginia, October 1, 1946.

"*Technique of the Test:* (1) Mark off a circular area, 6 cm. in diameter, in each antecubital area. Mark off all blemishes and marks in this area that might later be confused with petechiae. (2) Place a standard blood pressure cuff about each arm, and maintain in each a pressure of 35 mm. of mercury for fifteen minutes. Lower the pressure, and count and mark all petechiae within the two circular areas, using a good light and a magnifying lens of 5D or its equivalent. (3) One hour or more later, repeat, using a cuff pressure of 50 mm. of mercury."

The Petechial Index is calculated as follows: "To the number of petechiae occurring at 35 mm. of mercury multiplied by 2, add the additional number occurring at 50 millimeters. Based upon the Petechial Index, capillary fragility is considered to be: (a) normal, if the Index is 8 or less, (b) increased (abnormal) if the Index is 13 or more, and (c) borderline, but probably abnormal, if the Index is 9 to 12. In order to save time, the second stage can be omitted under the following conditions: (1) The number of petechiae after the first stage is 2 or less. Such persons may be considered normal. Usually, but not invariably, the person is normal who has 3 petechiae after the first stage. (2) If 6 or more petechiae appear after the first stage, the subject may be considered abnormal. (3) The test is a repetition, and may be compared with the corresponding first stage of an earlier test. Repetition in less than three weeks, however, is unreliable in any case. The second stage should always be done if the fragility is being tested in a subject for the first time and there are four or five petechiae after the first stage. It should also be done in most cases when the number after the first stage is three."

The thirteen patients in this series were selected from a total of sixty over a period from five to sixteen months, each of which had, in addition to complete medical, neurological, and laboratory examinations, a determination of the capillary fragility as evidenced by the aforementioned method. Only those patients that showed an increased or borderline capillary fragility were chosen for actual rutin therapy.

DOSAGE

The usual dosage was 20 mg., orally, t.i.d. Refractory cases may require 40-60 mg. t.i.d. to restore capillary fragility to normal.

RESULTS

Of the thirteen patients in this series (see tabulated chart) eight were marked hypertensives. Each of these patients showed increased capillary fragility, the indices ranging from twelve to twenty-two. Repeat determinations of the capillary fragility were made approximately every fourth week following the initial rutin therapy. Using a dosage of 20 mg. t.i.d., three patients showed a normal petechial index at the end of four weeks, one at the end of eight weeks, and another at the end of twelve weeks. Of the three remaining patients in this group the dosage of rutin was increased to 40 mg. t.i.d., with a resulting reversion in one case of the Petechial Index to normal at the end of sixteen weeks. Capillary fragility was not effected by treatment and remained abnormal in the other two in spite of respective rutin dosages of 180 mg. daily. Curiously enough, one of the latter two patients had an initial doubtful or bordering Petechial Index that became more definitely positive following treatment; this patient eventually made a lethal exodus as the result of renal insufficiency. Of the above six patients whose fragility returned to normal, all remained so with the continuance of the drug. In one instance rutin was deliberately discontinued on one of the above patients whose capillary fragility, as measured by Göthlin's test, had become normal and there was a reversion to a positive index. The drug was administered again and the fragility returned to normal. There was an insignificant fall in blood pressure in three of the above group; five were uninfluenced. Four patients evidenced retinal hemorrhages of minor degree; three hemorrhages subsided, and one persisted but did not become greater under rutin therapy. Prior to the institution of rutin therapy one patient complained of dizziness; another, of dizziness accompanied with headache. The symptoms became more intense with administration of the drug but could not definitely be attributed to rutin.

Because of the wide usage of thiocyanate medication in hypertensives and the tendency of this drug to produce cutaneous ecchymoses and retinal hemorrhages,[5] three of the group whose Göthlin Index had returned to a normal level were given potassium thiocyanate together with the rutin and the capillary fragility remained normal. Two patients from a sub-group with hypertensive arteriosclerotic heart disease and showing an initial normal capillary fragility were given no other medication except potassium thiocyanate and eventually one evidenced

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FIG. 2

DISEASE	PETECHIAL INDEX					BLOOD PRESSURE		RETINAL HEMORRHAGE		Evidence of Toxicity	Evidence of Sensitivity	Rutin Dosage	Thiocyanate Medication
	Normal at Onset	Positive at Onset	Unchanged	Return to Normal	Number Weeks	Fall	Unchanged	Persisted	Subsided				
Arterial Hypertension													
Case No. 1		x		x	4		x			None	None	20 mg. t.i.d.	x
Case No. 2		x		x	4	x			x	Dizziness (?)	"	20 mg. t.i.d.	
Case No. 3		x		x	12		x			None	"	20 mg. t.i.d.	x
Case No. 4		x	x	x	16	x		x		Dizziness Headache (?)	"	40 mg. t.i.d.	
Case No. 5		x		x	4		x		x	None	"	20 mg. t.i.d.	x
Case No. 6		x	x				x			None	"	60 mg. t.i.d.	
Case No. 7		x		x	8		x			None	"	20 mg. t.i.d.	
Case No. 8		Doubtful	x			x			x	None	"	60 mg. t.i.d.	
Hypertensive Arteriosclerotic Heart Disease													
Case No. 9	x*						x			None	"	20 mg. t.i.d.	x
Case No. 10	x					x				None	"	20 mg. t.i.d.	x
Recurrent Cerebral Arteriospasm													
Case No. 11		x		x	4		x			None	"	20 mg. t.i.d. Ascorb. Ac	
Secondary Anemia (Undetermined) Case No. 12		x		x	4		x			None	"	20 mg. t.i.d.	
Esophageal Hem. (?) Case No. 13		x		x	4		x			None	"	20 mg. t.i.d.	

*Became positive with thiocyanate therapy; returned to normal with rutin therapy.

an increased capillary fragility and soreness of several finger tips. The fragility became normal with the addition of rutin to the regimen and the finger discomfort disappeared. The other patient was given potassium thiocyanate for four months without disturbing the normal capillary fragility.

One patient with recurrent cerebral arteriospasm has, to date, shown promising results with rutin therapy.

CASE REPORTS

E. A., a sixty-eight year old white widow, was in general good health until June, 1945, at which time she "had a convulsion and became unconscious". Her LMD held very little hopes for her recovery. She was seen in consultation shortly afterwards because of a grossly irregular cardiac rhythm and bradycardia. Electrocardiographic evidence revealed a complete heart block, idioventricular rhythm and recent progressive myocardial degeneration. The auricular rate per minute was fifty; ventricular, forty-two per minute. Her course was uneventful and she did well until November 28, 1945, when she was admitted to the hospital because of a "convulsion". The outstanding physical findings on admission revealed the patient to have a generalized convulsion, worse on the left, with muscular weakness of the left face, arm, and leg. A diagnosis of cerebral embolus (rt.) was made at this time. Auricullar fibrillation was present and patient was digitalized. She was discharged via ambulance on December 5, 1945, seven days later, and patient was seen at her home shortly afterward at which time a physical examination revealed a fairly well developed and nourished female in no apparent distress and not acutely ill. Routine blood work was reported within normal limits. Ophthalmoscopic examination revealed normal retinal vessels and disks. With the exception of atelectatic rales in both bases the lungs were normal throughout. The apex beat was visualized slightly outside the left midclavicular line and there was cardiac dullness 1½ to 2 cm. to the left of the left midclavicular line. The rhythm was irregular; the rate was 45 per minute at the apex. The first and second sounds were of fair quality and a soft blowing systolic murmur was audible at the apex. B.P. 145/90. A neurological examination was essentially negative. Other physical findings were non-contributory. The evanescence of the hemiplegia following so shortly in the wake of the convulsion made one question the diagnosis of a cerebral embolus and favor one of cerebral arteriospasm. Because of the bradycardia one wondered if an underlying Stokes-Adams syndrome could not be the cause of the "convulsion". These thoughts were dispelled two months later (February, 1946) when patient complained of nausea, thickness of speech, and twitching of face. She was seen immediately and it was obvious that a convulsion was imminent. There were initial oscillations of the eyes, twitching of the left side of face, left arm and leg and soon a Jacksonian type convulsion. Right cerebral pathology was obvious. Examination of the heart revealed the first and second tones to be of fair quality. It was interesting to note that the rhythm was observed for the first time, to be perfectly regular; the rate was 80 per minute at the apex. This certainly did not look like a Stokes-Adams syndrome. Within forty-five minutes of the onset, the convulsions became generalized and so severe as to shake the bed. In spite of adequate sedation the convulsions and loss of consciousness lasted eight hours at the end of which time there was a typical left hemiplegia and dysarthria. A neurological examination revealed diminished reflexes on the left but no sensory disturbances. Within seventy-two hours there was little if any residual paralysis and the speech was perfectly normal. Patient made an uneventful recovery, but physical examination revealed that the cardiac rhythm had again become irregular and the rate was 40-45 per minute at the apex. One month later she experienced another episode with an identical course and convalescence. Intramuscular injection of 7½ gr. sodium amytal controlled the convulsions.

Because of the increasing frequency and severity of these attacks and because the underlying pathology possibly could be explained on the basis of cerebral arteriospasm or capillary rupture with intimal hemorrhage, capillary fragility (Göthlin's method) was determined with a resultant abnormal Petechial Index of twenty. On April 1, 1946, rutin 20 mg. t.i.d. was instituted and four weeks later there was a reversion of the capillary fragility to normal. There was some question as to an existing vitamin C deficiency and so ascorbic acid 100 mg. b.i.d. was administered in conjunction with the rutin.* Routine determinations for the following four

*It has been reported that the effectiveness of rutin is diminished in the presence of vitamin C deficiency.[7]

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months found the fragility normal and patient asymptomatic. One week before she was to have had her fifth fragility determination patient was said to have had a "mild" convulsive seizure. Patient was not observed until the following day, during which time she evidenced only slight residual paralysis of the left arm. Capillary fragility was determined at this time and found to be increased. Patient admitted that she had "run out" of rutin two weeks prior to the episode and failed to renew her supply. Administration of the drug was begun immediately and three weeks later the fragility was again normal. There have been no subsequent attacks.

Shanno[9] reported two cases of pulmonary hemorrhages of undetermined origin that responded to rutin therapy. In both instances the hemorrhages ceased and previously positive Göthlin Indices returned to normal. We were confronted with a problem of bleeding of undetermined origin from the upper alimentary tract (Case No. 13) that appeared to respond to the drug rutin.

CASE ABSTRACT

P. C., a white housewife, aged eighteen, complained of "vomiting" of small amounts of bright red blood, especially at time of periods (but not consistently) for a period of two years. Her last menstrual period occurred six weeks prior to her admission and patient was referred by her obstetrician, who believed correctly that she was pregnant, because of peculiar vicarious-like bleedings from the throat. She gave a history of having been kicked in the abdomen at the age of twelve years and of a gnawing epigastric pain (relieved by food) for a period of five years. The physical examination was non-contributory with the exception of suggestive epigastric tenderness on palpation. Laboratory studies included a red blood cell count of 3,600,000 per cu. m.m. with 72 per cent hemoglobin; white blood count and differential were normal, as well as the bleeding and clotting times. Laryngoscopic, fluoroscopic and x-ray examinations of esophagus, stomach and duodenum did not reveal any evidences of pathology. The lungs were clear. A P. I. (Petechial Index) of seventeen returned to normal within four weeks under rutin therapy and there have been no recurrences of the above episodes.

It is not at all unlikely that rutin may prove to be of value in the treatment of certain of the secondary anemias of undetermined origin.

The following case is one dealing with an anemia of undetermined origin observed early in pregnancy (six weeks) and complicated by a pot-pourri of signs and symptoms. It is presented with tongue in cheek for the efficacy of rutin in this particular instance is open to question in spite of the evidence of increased capillary fragility and the dramatic response to normal with rutin therapy.

CASE REPORT

E. D., an eighteen year old white married woman, gave as her chief complaint sharp pain in the left lower quadrant, of twenty-four hours duration. She gave a history of regular menses until the present, at which time there was an amenorrhea of six weeks duration. She stated that she had been in previous good health. Physical examination revealed a markedly anemic but well developed female lying restlessly in bed. She was obviously acutely ill. Temperature was 103°; pulse 110; respiration 26. There was only a suggestive stiffness of the neck and other neurological findings were not pathological. The lungs were clear, the heart normal in size but a transient soft systolic murmur was audible at the apex. Blood pressure 130/80. Most of the attention was centered at the left lower quadrant where there was moderate tenderness but no rigidity on palpation; the left flank was acutely tender on palpation and patient gave a history of having had previous renal pathology. No recent G.U. history was elicited. Sulfadiazine was ineffectual after twenty-four hours in influencing the course of her temperature and the picture was so suggestive of an ectopic pregnancy, in spite of the apparent generalized systemic infection, that an obstetrician was asked to see her in consultation. A blood work-up at this time revealed the hemoglobin to be 37 per cent; R.B.C. 4,410,000 per cu. m.m.; leukocytes 30,500 per cu. m.m.; PMN 90 per cent; lymphocytes 10 per cent. We decided to observe patient carefully. She was seen six hours later and because of such neurological manifestations as severe headache, suggestive stiff neck and fever of 104° she was admitted to St. Luke's Hospital. Fortunately, we located penicillin and adequate I.M. injections were begun immediately. Twelve hours later the blood examinations were repeated and the hemoglobin was reported as 36 per cent; R.B.C.

3,340,000; color index .54; leukocytes 10,300 per cu. m.m.; PMN 84 per cent; lymphocytes 14 per cent; large mononuclears 1 per cent; mast cells 1 per cent. Coagulation time was three minutes; bleeding time less than one minute. In regard to blood cell fragility, initial hemolysis began at .42 per cent. The heterophile antibody agglutination test showed agglutination in dilution 1:32. A voided urine specimen was reported to contain a distinct trace of albumen and fifteen to twenty pus cells per h.p.f. A spinal puncture was made without difficulty and the fluid was not under increased pressure. 248 red cells and one white cell were present per cu. m.m.; protein 25 mg.; sugar 60 mg. and chlorides 643 mg. per 100 c.c. A neurological examination at this time was non-contributory. Because of the reduced R.B.C. and hemoglobin, and amenorrhea and pelvic findings suggestive of an ectopic pregnancy (left) patient was admitted to surgery. At operation the abdomen was entered through a left rectus incision and no blood was encountered. Exploration of the cavity showed the uterus to be anterior and enlarged to the size of a six weeks pregnancy. The liver and spleen were normal in size and appearance. Both ovaries were cystic but for fear of aborting the patient these were left alone. Patient was returned to her room in fair condition. All subsequent laboratory procedures were reported negative. This included a repeated spinal puncture on the first post-operative day, spinal, blood and stool cultures, agglutination, serology, and an intravenous pyelogram. X-ray examination of the chest revealed peculiar breast shadows but this was afterwards explained on the basis of the pregnancy. The temperature returned to normal on the fourth post-operative day and after several 500 c.c. whole blood cell transfusions (positive Rh determination) she made an uneventful recovery and was discharged fourteen days following admission. She was seen one week later at which time there was a definite fall in the hemoglobin content in spite of iron and liver therapy. The capillary fragility was determined by Göthlins method. This was increased as evidenced by a Petechial Index of sixteen. Rutin, 20 mg. t.i.d., was administered and one month later the fragility was normal and there was an improvement in the hemoglobin content of 10 per cent. Unfortunately, the patient was not seen again for a period of five months during which time she did not receive rutin medication. Capillary fragility and blood picture taken at this time were within normal limits. One wonders whether the anemia could not have been influenced in the proper direction by the initial rutin therapy.

COMMENT

Rutin is a non-toxic drug related to hesperidin both in its structural arrangement and its ability to restore increased capillary fragility to normal. We are cognizant of the handicaps in evaluating the drug at this time. First, the series is too small. Secondly, the importance and relationship of the role of hypertension with or without increased capillary fragility as the cause of vascular accidents has not been definitely established. Further, the method used in determining the Petechial Index is crude and leaves much to be desired. Finally, very recent observations on the relationship of increased capillary fragility and increased cutaneous lymphatic flow[10] may throw more light on the choice of patients and the efficacy of rutin therapy.

SUMMARY

1. The results of a series of thirteen cases have been reported showing the effect of rutin, a comparatively new drug, in establishing normal capillary fragility.
2. Two cases have been discussed in which potassium thiocyanate was administered to patients showing normal capillary fragility with varying results. One patient developed an increased capillary fragility following continued dosage of the drug together with tenderness in several finger tips; rutin restored the fragility to normal and the finger discomfort disappeared. Another patient received thiocyanate indefinitely without increasing the fragility or causing untoward symptoms.
3. The use of thiocyanate in the treatment of hypertensive arteriosclerotic heart disease is not without danger once the patient shows evidence of increased capillary fragility. The continued use of the drug is apparently harmless in patients whose capillary fragility remains normal. Once the capillary fragility is returned to normal with rutin, thiocyanate may be used as an adjunct indefinitely without increasing the fragility.
4. By its ability to restore increased capillary fragility to normal, rutin may prove to be a valuable drug in such patients in preventing or in-

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hibiting cerebral vascular accidents and insidious bleedings of undetermined origin.

5. Of ten hypertensive patients evidencing increased capillary fragility and receiving rutin therapy, the blood pressures of seven were unchanged; three insignificantly lowered.
6. The retinal hemorrhages of three out of four patients subsided following administration of rutin.
7. The drug does not appear to have any toxic effects in man.
8. Cases refractory to rutin are occasionally encountered, in which event the usual dosage of 20 mg. orally t.i.d. should be increased to 40-60 mg. t.i.d., at least in some instances, before an increased capillary fragility may be expected to return to normal.

CONCLUSION

We must emphasize that this is merely a preliminary report, but from our observations we are of the opinion that (1) rutin may prove to be a valuable drug in preventing cerebral vascular accidents, retinal hemorrhages, and insidious bleeding of undetermined origin in those patients evidencing increased capillary fragility and responding to the drug. (2) Thiocyanate and related drugs may be given together with rutin indefinitely and without danger once capillary fragility has returned to normal. (3) The effectiveness of rutin in lowering the blood pressure is questionable.

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Tumor of Retina is Hereditary.

Retinoblastoma, a tumor of the retina, the membrane of light perception in the eye, is hereditary, according to Harold F. Falls, M.D., of Ann Arbor, Mich.

Writing in the January 18 issue of *The Journal of the American Medical Association,* Dr. Falls points out that both eyes were affected with retinoblastoma in female identical twins. The condition appeared at approximately six months of age in both. Because the parents refused either surgery or x-ray treatment for the pair, a fairly rapid extension of the tumor within the skull led to the death of both children.

Dr. Falls states that "it is my sincere conviction that parents producing any child with retinoblastoma should be strongly urged to stop all child bearing. It is also advocated that any person surviving enucleation [removal of the eyeball] for retinoblastoma be sterilized."

NEUROSURGICAL COMPLICATIONS OF INJURIES ABOUT THE HEAD AND FACE*

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Complete accurate appraisal of a patient's condition suffering from traumatic wounds of any type about the head cannot be definitely determined until a neurological examination has been done. Many times the patient is unconscious or uncooperative for various reasons which delay the early completion of such an examination. However, several gross tests can be carried out which are of great help. When there is an injury about the head or face, associated with an abrasion, laceration or fracture of the ipsilateral shoulder, one should always examine the cervical spine carefully, because stretching of the head shoulder angle may cause fracture of the laminae or articular facets of the spine. Often oblique rays are necessary to demonstrate the fracture. Meredith and Grantham called attention to this syndrome and cautioned about the examination if the patient complained of any stiffness or pain in the neck. It is important to know the power of motion in all extremities, and, if there is weakness of an arm or leg, to note carefully if the weakness progresses. This is especially true if the patient is drowsy and there is suspicion of an intracranial localized blood clot. In the drowsy or unconscious patient, pinching the arm or leg or sticking it with a pin will often cause motion when such motion cannot be elicited otherwise. Again, in the unconscious patient observation of the exposed chest and abdomen will often show characteristic movements, thereby aiding in diagnosing fracture dislocation of the cervical spine with complete physiological or anatomical disruption of the spinal cord. Inspection shows absence of chest motion and jerky excursion movements of the abdominal wall. During inspiration the abdominal wall rises and on expiration it falls rapidly. In addition, there is a fluttering like motion of the anterior abdominal wall at the end of expiration, showing absence of any muscular tone. The picture is quite characteristic. When deep lacerations are present about the cheeks, having the patient show his teeth, or, if unconscious, painful pressure over the supra-orbital nerves will cause movement about the mouth, showing that the facial nerve in question is intact. If facial wounds require dressings which cover one or both eyes for several days, the condition of vision in each eye should be tested, preferably by reading, but otherwise by counting the examiner's fingers with each eye before the dressing is applied. When unconscious, a light is flashed into the eyes and, if the pupils contract, sight is most likely present.

Careful inspection of the nares and external auditory canals should be made for the presence of cerebrospinal fluid leak. Cerebrospinal rhinorrhea occurs following fracture through the accessory nasal sinuses in the floor of the anterior fossa, tearing the cerebral dura, and fluid escapes from the subarachnoid space. It is difficult to tell if fluid is escaping when the patient is lying down as the fluid runs down the posterior nasopharynx. The patient is held in the sitting position with the head bent forward, thus allowing the escape of fluid through the nose. Generally blood from facial wounds will be found partially clotted in the external canal and at first glance it is impossible to determine if subarachnoid fluid is escaping from the ear. The canal should be cleaned gently with a cotton applicator, and then by turning the suspected ear downward fluid will escape if such a lesion is present. Should a fracture of the mandible be present, always suspect a basilar skull fracture, or cerebrospinal fluid leak from the ear, and examine the status of hearing in both ears.

Doctors are constantly amazed by the alertness and good general condition of an average patient exhibiting large ragged wounds of the face or scalp, often with facial bones crushed or actually missing or a comminuted skull fracture with actual extrusion of pulpified cortical tissue. In the recent war the severity of the brain injury was not always appreciated when only a small laceration 5-10 millimeters was found on the scalp from a high velocity fragment. However, the patient would be unconscious and die in a very short time. If autopsied the brain showed many diffuse and conglomerate petechial hemorrhages, areas of frank hemorrhage

*Read before the Norfolk County Medical Society, Norfolk, Va., May 13, 1946.

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and cortical destruction. Coleman has shown that only 5 to 7 per cent of head injuries show shock if the injury is limited to the head. Therefore, if shock is present, one should always examine the patient for associated injuries, notably fractures of long bones, intra-abdominal or intra-thoracic injuries.

Injuries involving the scalp, skull or brain may be divided into recent and delayed, when speaking in terms of the time element for surgical repair. The nature of wounds, regardless of etiology, requires the same basic principles of treatment. These are: Control of hemorrhage, prevention of infection, and restoration of the tissue to its functioning state if possible. Only too often does a severe injury involve comminution of the frontal or ethmoidal cells, or orbit and fracture into the cranial vault, tearing the dura and causing severe damage to the adjacent cerebral cortex. When such wounds exist, the treatment necessitates great skill to make the cranial repair and restore normal facial contour.

It is well to know that the cause of death from head injuries is, first, direct injury to the brain, with pressure or without pressure; second, infection from scalp wounds or compound fractures; third, intracranial clots which may be extradural, subdural or intracerebral. Also, the indications for operation in head injuries are, first, as previously stated, to prevent infection; second, to arrest and control hemorrhage; third, to elevate depressed fractures; and, fourth, to occasionally do a temporal decompression to control increased intracranial pressure.

The treatment of recent wounds of the scalp, those less than six hours old, consists of first shaving the hair from about the wound for at least three inches. The skin about the wound is painted with tincture of merthiolate or other suitable solutions, after which it is infiltrated with 1 or 2 per cent novocain one-half inch from the margins of the wound. This is followed by copious irrigation of the wound with normal saline or sterile water until the wound is thoroughly clean. Generally this requires three to six thousand cubic centimeters of water if the wound is dirty. Next, the edges are debrided until the cut surfaces ooze bright blood freely. If careful inspection and palpation of the skull shows no fracture, the wound is closed tightly in layers with interrupted black silk sutures. The practice of dusting sulfanilamide powder into wounds is discouraged since adequate debridement and control of hemorrhage before suturing the wound will result in primary union. Sulfa crystals have only very questionable bacteriostatic action when applied locally. If placed in the wound excepting as a very fine film, wound healing is delayed because of the hard, bulky mass of non-absorbable material remaining between the wound edges. Its local use was entirely stopped during the war.

Penetrating wounds with compound depressed fractures or through and through gunshot wounds of the brain often leave comminuted fragments of bone and small pieces of metal driven deeply into the cortex. In these instances it is necessary to elevate the bone fragments, remove all accessible in-driven bone and metal and thoroughly irrigate the tract of the missile. Often it is possible to insert a soft rubber catheter along the entire tract, and wash blood clots and destroyed brain tissue out. More vigorous efforts should be made to remove in-driven dirty bone fragments than relatively clean metal because brain abscesses are more prone to develop about pieces of bone.

Sulfathiazole powder should never be used on the surface of the brain as it is very toxic and irritating. Horrox relates two patients who had generalized convulsions shortly after the application of the drug while the wound was still open. If the ventricle has been entered, it is well to put 25 to 50 thousand units of penicillin directly in the cavity. Next, the dura should always be closed tightly to prevent infection. If the tear in the dura is too large to suture, a satisfactory patch can be taken from the exposed pericranium or temporal fascia. Again, the skin should be closed in layers without drainage. Penicillin is found in very low concentrations within the cerebrospinal fluid when given by the intramuscular route, but maintains an adequate level when injected either directly into the ventricular system or the lumbar subarachnoid space. However, smaller doses are given in the 24 hour period than by muscle, usually 25,000 units twice daily. This quantity may have to be reduced if the patient complains of neuritic pains into the legs or develops severe headache. These unfavorable symptoms are usually due to an irritable fixative rather than to toxicity of the yeast.

Recent war experiences definitely demonstrated that

many initially untreated compound depressed skull fractures and or with laceration of the cerebral cortex could be treated later like early wounds of similar nature with primary healing. This was because the wounds were adequately debrided, closed tightly and given penicillin both by intramuscular and intrathecal routes. I have operated on untreated or improperly treated cases in this fashion from 5 to 11 days after injury, with resulting primary healing.

Any repair of skull defects should be delayed for six weeks to three months. At the end of that time the defect may be repaired for cosmetic reasons or because the patient complains of a giddy feeling when stooping over. Contrary to popular opinion, repair of a skull defect will not cure epilepsy, which, of course, is due to brain damage. There are several ways of repairing a skull defect. Following the first world war, Colman reported on the use of antogenous grafts taken from the external table of the skull close to the defect. This was a long, tiring, bloody operative procedure and, if completed, the pieces of bone often absorbed in several months, leaving the original defect. Later, bone grafts were taken from the shin or ribs, and seemed more satisfactory. Celluloid plates, containing many perforations fitted to the defect may be moulded while warm between two pans, placed in the defect and sutured. The procedure is very simple, and satisfactory results are obtained equal to other methods. Vitallium, and especially tantalum, has been used since the war frequently to repair defects. Tantalum is easily cut and moulded at the operating table, and is probably the most inert of the metals when buried in tissues.

One of the most deceptive and grave complications of skull fracture is an extradural hemorrhage. This lesion occurs when the fracture line crosses the middle meningeal artery groove, tearing the artery, and a localized extradural hemorrhage results. The classical diagnostic picture is an injury to the head, followed by increasing drowsiness and unconsciousness several hours later, dilatation of the ipsilateral pupil and contralateral hemiparesis, slow pounding pulse, rise in blood pressure, and occasionally generalized convulsions. The treatment consists of trephining the skull, controlling the hemorrhage and removing the blood clot. The chances of the patient recovering are directly proportional to the time the clot is removed following injury. Extradural hemorrhage in the posterior fossa is rare but does occur and requires prompt removal when cerebellar symptoms develop. Acute subdural clots are generally fatal because a major vessel is usually torn at an inaccessible place on the inferior surface of the brain and it is impossible to clamp the bleeding points. Acute intracerebral clots may be located anywhere in the subcortical tissue and often a ventriculogram will be necessary for localization.

Delayed lesions of the central nervous system following trauma must always be kept in mind and proper treatment begun when any are suspected. Chronic subdural hemorrhage may give symptoms several weeks or months following even a very trivial injury. It is generally easily removed by placing a burr hole over the suspected clot and removing the liquefied, greenish brown fluid. Burr holes should always be made bilaterally, as about 25 per cent of the clots occur on both sides of the head. Pneumocephalus may appear rapidly or be delayed for several days following a fracture of the frontal, ethmoid or sphenoid sinus with tearing of the dura, which allows air to escape into the cranial cavity and become pocketed. These lesions are more frequent following gunshot wounds of the sinuses than from blows about the head which cause fracture. Surgical repair of the dura and aspiration of the air by utilizing an appropriate osteoplastic flap is the treatment of choice.

Gas gangrene of the head and face are fortunately very rare because of the good blood supply of these tissues. However, because of the close proximity to the central nervous system this infection approaches 100 percent mortality.

When infection of the scalp does occur, any wound should be opened widely and warm wet dressings applied every four hours. Enough sulfadiazine is given by mouth to keep the blood level at about 10 mg. per cent and 25,000 units of penicillin is given intramuscularly every three hours. An infected scalp, whether in the location or presence of a skull fracture is a very serious lesion. This is because there are many thrombosed infected veins in the infected laceration which can extend through the diploe of the skull, resulting in osteomyelitis of the skull, extradural abscess, subdural abscess, meningitis, or brain abscess. All of these lesions with the exception of meningitis and possibly subdural abscess require surgical intervention and all are

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serious complications which may result fatally.

Cavernous sinus thrombosis occurs from infection about the upper portion of the face and is brought to the sinus by infection in the angular and smaller veins about the eyelids. This lesion is also quite characteristic, as shown by chemosis, proptosis, varied extra-ocular palsies, engorgement of the retinal veins and early papilledema, with evidence of facial infection. In spite of the use of the sulfonamide drugs, penicillin and general measures directed toward treating the infection, the mortality rate is still high.

Surgical attack on the cranial nerves following traumatic injury, with the exception of the facial nerve, is not only discouraging but generally useless. Occasionally a displaced fracture line of the floor of the anterior fossa compresses or partially severs the optic nerve by narrowing the optic foramen and edema of the nerve occurs which will destroy the remaining nerve fibers. If such a condition exists, the optic foramen may be decompressed through an osteoplastic flap. Contusions, lacerations and fractures of the face frequently destroy one or more of the three peripheral sensory branches of the trigeminal nerve that cause numbness of a portion of the face for several months. Excepting for the subjective uncomfortable feeling of numbness there are no ill effects of the lesion. The patient should be reassured that the feeling will return in a few months.

The anatomy of the facial nerve is such that a functional repair in the bony canal and close to the muscular innervations on the face is very difficult and many times impossible. Primary end to end suture of the nerve is the repair of choice. However, varied degrees of mass motion of the face and diminution of natural facial expression always results from the best facial repair if complete severance of the nerve has occurred. Nerve grafts are generally unsatisfactory. Facio-hypoglossal or facio-accessory anastomosis are often done when the nerve is destroyed close to or within the bony canal, making approximation impossible. Should enough time elapse following injury to the facial nerve so that fibrosis of the facial muscles has occurred, it then is necessary to use a mechanical method for restoring balance to the face. Brown has devised fascial slings placed in the subcutaneous tissue of the face from the lips to the temporal fascia and muscle that corrects this deformity satisfactorily. Needless to say, only slight motion and no facial expression can result from this procedure.

Finally, mention should be made of the general treatment of the patient with cerebral concussion or contusion. The head of the bed should be elevated 8 to 12 inches, which allows for better venous drainage from the head and also tends to reduce the intracranial arterial pressure. In this position, should the patient develop fluid in the lungs, as they often do if drowsy, lower the head of the bed, raise the foot sharply, turn the patient on his abdomen and his head to the side. Often several ounces of fluid will escape in a few minutes' time. The procedure is often life saving. Generally morphine should be avoided, but if the patient has severe associated injuries for which morphine is indicated and close observation of the patient is possible, appropriate doses may be given. Only about 3 to 5 per cent of these patients have intracranial hypertension of severe enough degree to restrict fluids, so an intake of 1,800 to 2,500 c.c. in twenty-four hours should be encouraged. Generally no dehydrating drug is given unless the patient becomes worse from intracranial pressure. Lumbar punctures are used for diagnosis of subarachnoid hemorrhage and occasionally for reducing pressure, but certainly it is not a procedure to be used on all patients with head injuries. Excepting when immediate operation is planned, the x-ray examination of the skull may be delayed until such time when the patient is cooperative, and will tolerate transportation to and manipulation of the head in the x-ray department.

405 Wainwright Building.

DIABETIC ACIDOSIS
A Review of 105 Cases*

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There is no standardized treatment of diabetic acidosis. Differences are found in reports of Joslin, *et al*,[1] Almy *et al*,[2] Peters,[3] and others. The chief points at issue are (1) the dosage of insulin, (2) the early use of glucose, and (3) the advisability of using alkalis, such as 1/6 molar sodium lactate solution intravenously. This report is based on a review of 105 cases of diabetic acidosis treated on the medical service of the University of Virginia Hospital from January 1, 1939, to September 1, 1945.

In this period under study, Table I, there were 71,965 admissions to this hospital. Diabetic acidosis was present in 105 (7.69 per cent) of 1,366 diabetic patients. A carbon dioxide combining power of

TABLE I
DIABETIC ACIDOSIS
January 1, 1939—September 1, 1945
Definition: CO_2 Combining Power—35 Vols. % and below

1. Total hospital admissions		71,965
2. Total Diabetic admissions (including acidosis)		1,366
3. Percentage admissions for diabetes		1.89%
4. Total cases of acidosis		105
5. Deaths		8
6. Mortality Rate		7.62%
A. Uncomplicated cases	3	2.76%
B. Complicated cases	5	4.86%

the plasma of 35 volumes per cent and below has been chosen arbitrarily as the criterion for the diagnosis of diabetic acidosis. There were 8 deaths in these 105 patients. Thus, the crude mortality rate was 7.62 per cent. Three, or 2.76 per cent, were believed to be due to diabetic acidosis *per se*, and 5, or 4.86 per cent, occurred in cases with complications thought sufficient in themselves to cause death without the added factor of acidosis.

SEVERE ACIDOSIS

A carbon dioxide combining power of the plasma of 20 vols. per cent or below has been regarded by many writers as indicative of more critical and severe acidosis. A graver prognosis is said to be associated with a state of unconsciousness or "coma" than with a condition of semi-consciousness or drowsiness. In this series, there were 64 cases of severe acidosis. Five of these (7.81 per cent) terminated fatally, as shown in Table II. Twenty-one (32.78 per cent)

TABLE II
SEVERE DIABETIC ACIDOSIS
CO_2 Combining Power 20 Vols. % and Below
64 Cases

1. Crude Mortality rate	5 deaths	7.81%
2. Condition on admission to hospital		
A. Comatose	21 cases	32.78%
B. Semicomatose	43 cases	67.19%
3. Mortality rate according to condition		
A. Comatose	3 deaths	14.25%
B. Semicomatose	2 deaths	4.65%

were admitted in coma and 3 of these (14.25 per cent) died. Forty-three (67.19 per cent) were semicomatose and 2 (4.65 per cent) died. Thus, the mortality rate in this severe acidosis group increased approximately three times when the patient was in coma.

The cause of the 8 deaths, only 3 of which were believed to result primarily from diabetic acidosis, are analyzed in Table III. In 6 of these fatal cases,

TABLE III
FATAL CASES OF DIABETIC ACIDOSIS

Age	*Sex*	*Insulin Before Admission*	*Hours Duration of Coma*	*Initial CO_2 Vols. %*	*Complications*
43	Female	None—5 days	10	18.2	None
50	Male	None—4 days	30	23.1	None
15	Male	Yes (?)	6*	15	None
14	Female	None	24	12.7	Lobar pneumonia with effusion
78	Male	None	6	8.5	Coronary thrombosis
75	Female	?	12*	33.4	Generalized peritonitis
64	Female	None—2 days	18	24.1	Cerebrovascular accident
31	Female	None—2 days	24	13.8	Subarachnoid hemorrhage

*Semicomatose

*From the Department of Internal Medicine, University of Virginia Hospital.

the patient had either never taken insulin or had discontinued insulin treatment. Five were over 40 years of age. Four were over 50 years of age and had an average age of 66.7 years. The average age in the 3 fatal cases due to acidosis alone was 36 years. The complications thought to be the cause of death in 5 cases were respectively: lobar pneumonia with pleural effusion, coronary thrombosis with myocardial infarction, generalized peritonitis, cerebrovascular accident, and subarachnoid hemorrhage.

PRECIPITATING CAUSES

Precipitating causes of diabetic acidosis, as shown in Table IV, conformed to those in previous reports.

TABLE IV

PRECIPITATING CAUSES OF DIABETIC ACIDOSIS

1. Failure to take insulin as instructed		34
2. No known cause:		
A. Undiagnosed diabetic	14	
B. Treated diabetic	12	
	—	26
3. Common cold		11
4. Dietary indiscretion		6
5. Furunculosis		6
6. Lobar pneumonia		5
7. Pulmonary tuberculosis		4
8. Cellulitis		2
9. Empyema		1
10. Purulent otitis media		1
11. Atypical pneumonia		1
12. Subarachnoid hemorrhage		1
13. Coronary thrombosis with infarction		1
14. Other causes		6
		105

Failure to take insulin as instructed was responsible in 34, about one-third, of the cases. No cause for acidosis could be found in 26 patients. There was no history of diabetes in 14, and 12 were being treated for diabetes when acidosis developed. The common cold was thought to be the precipitating factor in 11 cases. Miscellaneous causes included dietary indiscretions, furunculosis, cellulitis, lobar pneumonia, empyema, purulent otitis media, atypical pneumonia, subarachnoid hemorrhage, and coronary thrombosis with infarction.

A brief analysis of the 8 fatal cases is given below:

Case 1.—F. W., A 43-year old colored female admitted January 28, 1940, voluntarily discontinued her insulin for 5 days and gradually lapsed into coma 10 hours before admission. The urine sugar, acetone, and diacetic acid were 4 plus. The carbon dioxide combining power of the plasma was 18.4 vols. per cent, and the blood sugar was 454 mg./100 cc. The patient was given 4,000 cc. of intravenous normal saline and 140 units of regular insulin during the first 3 hours. Later, 1,400 cc. of 1/6 molar sodium lactate solution was given intravenously. Within 15 hours, the ketonuria had cleared and the carbon dioxide combining power of the plasma had risen to 43 vols. per cent. However, she remained unconscious and died several hours later. A total of 375 units of regular insulin was given. Death was believed to be due primarily to diabetic acidosis of long duration.

Case 2.—F. C., a 50-year old colored male admitted April 7, 1943, had had diabetes for 11 years but had never adhered to a diet or an insulin program. Four days before admission, he discontinued insulin. This was followed by epigastric pain and nausea with vomiting. Thirty hours before admission, he became unconscious. Tests for sugar and acetone in the urine were strongly positive. The carbon dioxide combining power of the plasma was 23.1 vols. per cent. The blood sugar was 421 mg./100 cc. Twelve hundred cc. of 1/6 molar sodium lactate solution, 3,500 cc of 5 per cent glucose in normal saline, and 1,500 cc. of normal saline were given intravenously. Two hundred units of regular insulin were given during the first 3 hours. In 8 hours, the urine acetone had been reduced to 1 plus and the carbon dioxide combining power of the plasma was 55 vols. per cent. In spite of this, he became anuric, remained unconscious, and died 2 hours later. A total of 360 units of regular insulin was given. Death was attributed to diabetic acidosis.

Case 3.—R. L., a 15-year old white male admitted July 12, 1944, had unregulated diabetes for many months but had not sought medical aid. He vomited for 1 day and became stuporous 4 hours before admission. Laboratory examinations showed a 4 plus sugar and 3 plus acetone in the urine, a blood sugar of 533 mg./100 cc., and a carbon dioxide combining power of the plasma of 15 vols. per cent. One hundred and seventy units of regular insulin, 1,500 cc. of normal saline, and 1,200 cc. of 1/6 molar sodium lactate solution were given during the first 3 hours. In 9 hours, the carbon dioxide combining power of the plasma had increased to 46.7 vols. per cent. However, he re-

mained stuporous and disoriented and became anuric 3 hours later. In 24 hours, although the acetone in the urine had been reduced to 1 plus, the patient developed peripheral vascular collapse and died. Death apparently resulted from prolonged acidosis.

Case 4.—R. E., a 14-year old white female admitted December 15, 1941, had had polyuria, polydipsia, and weight loss of 6 months' duration. She had marked nausea and vomiting for 3 days. She became unconscious about 24 hours before admission. Examination revealed lobar pneumonia with pleural effusion. The urine acetone was 3 plus and the sugar 4 plus. The blood sugar was 435 mg./100 cc. The carbon dioxide combining power of the plasma was 12.7 vols. per cent. Treatment consisted of 1,200 cc. of normal saline and 2,400 cc. of sodium lactate solution intravenously and 115 units of regular insulin during the first 3 hours. After 8 hours, 1,200 cc. of 5 per cent glucose was given intravenously. There was no ketonuria after 18 hours and the carbon dioxide combining power of the plasma had increased to 48.1 vols. per cent. Three hundred units of insulin were given during the first 24 hours. The acidosis did not recur but the patient became anuric. Death, which occurred 4 days later, was thought to be due to lobar pneumonia.

Case 5.—H. T., a 78-year old white female, was admitted June 20, 1943. She had been bedridden for 2 years with arthritis and was not a known diabetic. She had "felt bad" for 5 days and was found in coma 6 hours before admission. The urine sugar test was 4 plus and the acetone and diacetic acid tests were 1 plus. The carbon dioxide combining power of the plasma was 8.5 vols. per cent. The blood sugar was 400 mg./100 cc. Treatment consisted of 1,800 cc. of intravenous sodium lactate solution, 1,000 cc. 5 per cent glucose in normal saline, and 200 units of insulin during the first 3 hours. About 2 hours after admission, the patient responded slightly to questioning. Irregular cardiac rhythm and a pericardial friction rub were then noted for the first time. An hour later, the carbon dioxide combining power had increased to 36.7 vols. per cent. The patient died suddenly 4 hours after admission. The cause of death was thought to be coronary thrombosis with myocardial infarction.

Case 6.—M. T., a 75-year old diabetic colored female admitted January 11, 1945, had had severe acute abdominal pain, nausea, and vomiting for 5 days. She was semi-conscious on admission. The urine sugar was 4 plus but there was no acetone. The carbon dioxide combining power of the plasma was 33.4 vols. per cent. There was marked spasm and tenderness in the right side of the abdomen, believed to be due to a generalized peritonitis following the rupture of a viscus. She was given 80 units of regular insulin and 4,000 cc. of 5 per cent glucose in normal saline. Six hours later, the carbon dioxide combining power of the plasma was 52 vols. per cent and the blood sugar was 129 mgs. per cent. The patient died 21 hours after admission, presumably from peritoneal infection.

Case 7.—J. B., a 64-year old white female diabetic, was admitted February 3, 1943. She became comatose 18 hours before admission and was thought to have had a cerebrovascular accident. The urine sugar was 4 plus and the acetone was 2 plus. The carbon dioxide combining power of the plasma was 24.1 vols. per cent. The blood sugar was 434 mg./100 cc. Fifty units of insulin were given during the first 3 hours and 160 units during the first 24 hours. Fifteen hundred cc. of normal saline and 1,200 cc. of 1/6 molar sodium lactate solution were administered intravenously. In 24 hours, the ketonuria had cleared and the carbon dioxide combining power of the plasma had increased to 47.8 vols. per cent. The patient remained comatose and became anuric on the third day. The acidosis did not recur. Death, which occurred 4 days later, was thought to be due to the cerebrovascular accident.

Case 8.—M. W., a 31-year old colored female diabetic, admitted April 8, 1943, was found unconscious 24 hours before admission. Repeated spinal fluid studies were consistent with a subarachnoid hemorrhage. The urine sugar and acetone were 4 plus. The carbon dioxide combining power of the plasma was 13.8 vols. per cent. The blood sugar was 382 mg./100 cc. Two hundred and ten units of insulin were given during the first 3 hours, together with 1,200 cc. of sodium lactate solution and 5,500 cc. of 5 per cent glucose in normal saline intravenously. In 12 hours, the ketonuria had cleared. Several hours later, the carbon dioxide combining power of the plasma was 42.7 vols. per cent. In spite of adequate control of the diabetes, she remained comatose and died 4 days later. Subar-

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achnoid hemorrhage was believed to be the cause of death.

Unfortunately, permission for post-mortem examination was refused in all of the cases.

TREATMENT

In this series of cases, treatment of *severe* cases of diabetic acidosis has conformed largely to the following program:

1. On admission, a urine specimen is obtained, the patient being catheterized if necessary, and tested at once for sugar, acetone, and diacetic acid. Blood samples are taken for sugar and plasma carbon dioxide combining power determinations. A laboratory technician is available for this work at all times.

2. Insulin.—Fifty units intravenously and 50 units subcutaneously (or more) of the quick-acting (regular) insulin are given initially and repeated in 50 unit doses at 30-45 minute intervals in an attempt to give about 300 units in the first 3 hours. Thereafter, insulin is given as indicated. These large doses of insulin are not advised as a routine procedure unless frequent urine sugar and blood sugar determinations are made. Otherwise, profound hypoglycemia may occur.

3. Fluids and Alkali.—An infusion of normal saline is started promptly and continued so as to supply 750-1,000 cc. of fluid per hour during the first 4-5 hours. During the first hour, 1/6 molar sodium lactate solution is given intravenously in 600 to 1,200 cc. amounts, usually the latter.

4. Urine specimens are obtained for testing every ½ hour if a catheter is in place or every hour if the patient is voiding.

5. The blood sugar and the carbon dioxide combining power determinations are repeated at the end of the first 3-5 hour period and again 6-8 hours later.

6. About 3-4 hours after treatment is started, glucose is given if there is indication of a decreasing urine or blood sugar. This may be by an infusion of 1,000 cc. of 5 per cent glucose in normal saline intravenously, *or* by sweetened water, orange juice, or milk, if the patient can take these fluids by mouth.

7. Cleansing enemas are not believed necessary nor do retention enemas seem beneficial.

8. The patient is allowed nothing by mouth if he is nauseated or unconscious. If the patient is not vomiting, gastric lavage is not indicated as this would serve only to remove a saline solution which the patient can use.

9. Small glucose feedings with supplementary insulin are given at frequent intervals (every 3-4 hours) until an adequate diabetic maintenance program is established.

10. Stimulants, blood transfusions, plasma, and other supportive measures are administered as needed.

DISCUSSION

Inasmuch as many of these patients came from a large, rural, mountainous area, it is probable that the cases of this condition observed on the medical service of the University of Virginia Hospital are in general more severe than those treated in large city hospitals. Frequently, the patient may be 12 or more transportation hours from the hospital at the time he becomes ill enough to seek hospital attention himself or his local doctor advises hospitalization.

The amount of quick-acting (regular) insulin used in the treatment of diabetic acidosis is arbitrary. However, there has been a trend toward the use of larger initial doses of insulin in recent years in our severe cases of acidosis. After January 1, 1944, an average of 255 units of insulin was given during the first 3 hours, which was approximately 100 units more than was used previously. This is shown in Table V. During this same time, the

TABLE V

SEVERE DIABETIC ACIDOSIS

CO_2 Combining Power 20 Vols.% and Below

1. Average amount insulin first 3 hours:		
A. (1-1-39 to 1-1-44)	45 cases	144 units
B. (1-1-44 to 9-1-45)	19 cases	255 units
2. Average amount insulin first 24 hours:		
A. (1-1-39 to 1-1-44)	45 cases	349 units
B. (1-1-44 to 9-1-45)	19 cases	450 units
3. Average fluid intake first 24 hours:		
A. (1-1-39 to 1-1-44)	45 cases	5976 cc.
B. (1-1-44 to 9-1-45)	19 cases	6881 cc.

average fluid intake was increased from 5,976 cc. to 6,881 cc., or about 900 cc. The largest amount of insulin given to any single case during the first 3 hours was 375 units and the largest amount in the first 24 hours was 830 units.

An alkali in the form of 1/6 molar sodium lactate solution has been used during the initial 3 hour period in approximately 2/3rds of our cases of *severe* acidosis. The remaining 1/3rd usually re-

ceived intravenous normal saline alone. The average carbon dioxide combining power on admission of the case in *severe* acidosis was 13.6 vols. per cent. The initial carbon dioxide combining power of the group of patients treated with sodium lactate was slightly lower (13.2 vols. per cent) than those in which an alkali was not used (15.6 vols. per cent). At the end of the first 12 hour period, the carbon dioxide combining power in those treated with sodium lactate was 47.7 vols. per cent, whereas in the group in which this form of treatment was not used, it was only 33.3 vols. per cent, as shown in Table VI.

TABLE VI
SEVERE DIABETIC ACIDOSIS
CO_2 Combining Power 20 Vol.% and Below—64 Cases

1. Average Initial CO_2	13.6 vols.%
A. Treated with normal saline	15.6 vols.%
B. Treated with sodium lactate	13.2 vols.%
2. Average CO_2 at the end of 12 hours:	
A. Treated with normal saline	33.3 vols.%
B. Treated with sodium lactate	47.4 vols.%
3. Average CO_2 at the end of 24 hours:	
Treated with sodium lactate	51.3 vols.%

At the end of the first 24 hours, the carbon dioxide combining power in those cases which received sodium lactate was 51.3 vols. per cent. In a report of another series in which an alkali was not used, it was noted that the carbon dioxide combining power was "12 vols. per cent initially; this had risen to 30 vols. per cent on the second hospital day, and on the third hospital day to 42 vols. per cent".[1] It has been argued that the use of an alkali "may actually be harmful and may divert attention from the truly important matter of giving enough insulin".[1] The use of sodium lactate has been favored in our series and we have not observed any harmful effects from its use. The patients treated with sodium lactate and saline received on an average a greater total sodium intake than those treated with saline alone. This was undoubtedly a factor in the more rapid rise of the plasma carbon dioxide combining power in the first group of cases. However, sodium lactate has been shown by other workers to be more efficacious in increasing the alkali reserve than sodium chloride because of the prompt liberation of the sodium ion. It is our impression that not only does the carbon dioxide combining power of the plasma increase more rapidly in the patients who are given sodium lactate solution, but also that clinical improvement occurs sooner, other factors being equal. In cases of *severe* acidosis with marked depression of the carbon dioxide combining power, we believe that it should be of considerable benefit to correct the acid-base balance to physiological levels in as rapid a manner as possible.

The early administration of glucose was not a part of the routine treatment of this series of cases, but it was used frequently after the preliminary large doses of insulin. Peters,[3] Almy *et al*,[2] and Conn and Bauer[4] offer convincing reasons for the early use of glucose. Glucose will prevent so-called "starvation diabetes", hypoglycemia and consequent prolongation of acidosis and ketonuria.

SUMMARY

1. One hundred and five cases of diabetic acidosis treated at the University of Virginia Hospital are reviewed. The crude mortality rate in this series was 7.62 per cent.

2. In 64 cases of severe diabetic acidosis, the crude mortality rate was 7.81 per cent. There were 3 (14.25 per cent) deaths in the 21 comatose patients, whereas only 2 (4.65 per cent) of the 43 semi-comatose ones died.

3. A program of treatment of diabetic acidosis is outlined.

4. Intravenous sodium lactate solution (1/6 molar) was used initially in two-thirds of the cases of severe acidosis. No harmful effects were noted and a more rapid rise of the carbon dioxide combining power of the plasma was obtained.

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EXPERIENCES OF AN OPHTHALMOLOGIST ABOARD A HOSPITAL SHIP*

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At the outset of this presentation let me say that I am not attempting in any way to deliver a scientific exposé, nor a statistical report of the work that is done by a hospital ship in the advanced theatres of war. Such matters are better left to more capable scientific and statistical minds than mine. It has occurred to me, however, that a report from one who was fortunate enough to have such duty might be of some interest to the members of this Society.

There are two main functions of a Navy hospital ship which are paramount above all else. These are to evacuate wounded personnel from the beachheads and to act as a floating hospital for the fleet in advanced areas. Subsidiary functions may be listed as: (1) To act as a medical supply depot for the other ships of the fleet; (2) To furnish library facilities for the medical officers of the fleet; and (3) To provide consultation facilities for other ships and for any shore based Army or Navy units that may desire them.

The personnel of such a ship is divided into the line and the staff, the latter consisting of the medical department and the supply department. In my own ship, the U. S. S. Relief, the medical department consisted of twelve regularly assigned medical officers, four dental officers, twelve nurses, and approximately one hundred and eighty hospital corpsmen. The medical officers included the senior medical officer, the chief of surgery with two other surgeons, the chief of medicine with two other internists, the otolaryngologist, the ophthalmologist, the urologist, the roentgenologist and the pathologist. In addition to these there were two surgeons assigned to the ship for temporary duty in each major campaign. Invaluable assistance was rendered on beachhead evacuations by the four dental officers who worked in the wards giving blood and plasma transfusions, administering anesthesia, and helping with dressings on patients.

Physically the ship was built much like any good hospital ashore with the exception that in the wards the bunks, which had very comfortable springs and mattresses, were either in two or three tiers. However, there was always enough space allowed so that the patients could be handled properly in their bunks. Operating facilities consisted of one main operating room with three tables ready for immediate use, a smaller operating room in the eye, ear, nose, and throat clinic with one table, and operating tables in dressing rooms off three of the larger wards. This gave us a total of seven operating tables which could be put to immediate use when casualties were brought aboard. With a medical complement such as ours the question will undoubtedly arise as to how we obtained assistance in the operating room. Most of this came from hospital corpsmen who were trained operating room technicians and who did a grand job in helping out.

In addition to the surgical set-up there were various other departments on the ship, such as a completely equipped x-ray department; a complete laboratory, including rabbits and guinea pigs for experimental purposes; a department of physiotherapy; the dental department with four operating chairs and a complete laboratory for the manufacture of dentures; spaces set aside for the taking of electrocardiograms and basal metabolic rates; and, last, but I think by no means least, the eye, ear, nose, and throat department.

In regard to the latter I should perhaps go into a little more detail as to the physical set-up. As I have stated before, there was a medium sized operating room with one operating table and two treatment chairs in the same room. We had a small office and a refraction range which, when closed off, made our dark room. In this we had the perimeter and slit-lamp. We were fortunate, and I think rather unique, in that we had both an ophthalmologist and an otolaryngologist. Until I joined the ship in the summer of 1943 these two departments had been combined, but it was found that there was enough work for two men and so I was sent out as the ophthalmologist. This worked out ideally, for during a good deal of the time, between invasions, we were engaged in servicing the fleet and, on such occasions, it was not unusual to run from fifteen to eighteen refractions a morning, with

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operations in the afternoon and consultations sandwiched in between. On the ear, nose, and throat side I have seen frequently as many as five tonsillectomies plus a submucous resection scheduled for the same morning. Our assistance in the clinic consisted of four hospital corpsmen who were trained in eye and in ear, nose, and throat work and who proved to be invaluable.

In connection with the above mentioned operating schedules in our department a word of explanation might be interjected at this point. One might well wonder why elective procedures were carried out aboard a hospital ship in the advanced theatres of war. When we were acting as a hospital for the fleet it was our policy to do anything that was required, just as we would have done had be been located in a hospital ashore. For instance, if the medical officer of a battleship felt that one of his crew was handicapped in the performance of duty by some bad tonsils, or a squint, the patient would be accepted aboard our ship for such surgery as was indicated. It stands to reason that such cases were not accepted when there was an invasion imminent or when there was a possibility that we would be moved.

The question of eye instruments presented an interesting problem at times and serves to show what can be done aboard such a ship with proper coordination. When I joined the ship the instruments for eye surgery, while adequate, were not all that was to be desired. Orders were immediately placed for additional instruments but due to the movements of the ship, and the rapid progress of the war, these orders never caught up with us. I found that we had no satisfactory means for localizing intraocular foreign bodies and, since I had been brought up with the Pfeiffer modification of the Comberg technic, I ordered such an apparatus. It did not arrive and I was forced to enlist the help of the dental department. One of the dental officers agreed to help me and we took a mold of a volunteer's eye and then the dentist made a transparent acrylic lens from the mold. Four small holes were drilled in this lens at the limbus at 12, 3, 6, and 9 o'clock and these filled with silver amalgam. This gave us a very satisfactory contact lens for localizations and proved invaluable in numerous instances thereafter.

Another instance of manufacturing instruments came in the quest for a handy knife for use in opening the sclera posteriorly in cases of intraocular foreign bodies. We had many of these cases and it seemed to me the Graefe knife was at best an unwieldy instrument for posterior incisions in the sclera. Consequently, I gave a muscle hook to one of the machinist's mates and asked him to work out on it. We finally emerged with a square blade about one millimeter on a side and with two cutting edges, one on the under surface and one on the inner surface of the blade. The shaft of the instrument from the blade to the handle was notched in millimeters so that one could tell fairly accurately how far posterior to the limbus he was placing the blade. This same machinist, although used to working on giant turbines and other large machinery, also modified a small forceps for me by fashioning two tiny cups on the blades. This was used for the extraction of non-magnetic foreign bodies from the vitreous in such cases where the foreign body could be seen with the ophthalmoscope.

The ship was designed to take care of approximately four hundred and fifty patients in the wards and rooms. All of these were located above the waterline with good ventilation by portholes and blowers. These accommodations were perfectly adequate when we were servicing the fleet, for in such a case we did not usually run more than three hundred patients aboard at any one time. For invasions of enemy territory, however, we realized that this number of beds would be inadequate and, consequently, on such occasions, the ward space was modified considerably. The crew moved out of their compartments and these were converted into wards, the crew sleeping in cots and hammocks on the main deck which was enclosed in canvas for the emergency. The entire promenade deck was also enclosed in canvas and cots placed on it throughout its length, this deck then being used for an additional large ward. These measures gave us a total of approximately seven hundred beds ready for use when we came into a battle area.

It might be thought that with a group of medical specialists aboard everyone stuck to his own specialty. This was the case at all times except when we were taking wounded aboard from the battle area. I have seen seven hundred badly wounded men come aboard within a space of a little over four hours and it must be obvious that in such a time there is no immediate place for specialization.

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Everyone pitches in and does what he can. Probably the most important immediate thing is the treatment of shock and every single person available lends a hand in this. My own ward in these instances had seventy-two bunks in it. In this I had all the primary eye cases, which would usually run about twenty-five to thirty, and the remainder of the space was taken up with every conceivable kind of wound. If major surgery was required these patients were made as fit as possible and sent to the main operating room. If minor surgery was all that was indicated, that was taken care of in the ward dressing room.

In an attempt to show the duties of this type of ship in her primary role, it might be well to try to describe our first mission of this type. Prior to the opening of the Marshall Islands campaign in late January, 1944, hospital ships had never been sent into a beachhead, but, in accordance with the Geneva Convention rules, stayed five hundred miles away from the battle zones. It is true that during the Gilbert Islands operations the only two hospital ships then in the Pacific, the Relief and the Solace, were ordered into the Gilberts but neither one of us went to Tarawa, where the fighting was heaviest. We hove to off the island of Apamama and took casualties that transports brought out to us. However, with the opening of the Marshalls offensive we went into the beaches for the first time.

During the night of D Day in the Marshalls we were heading for Kwajalein, steaming alone and fully lighted, up the middle of a channel with Japanese held islands on either side. Although nothing was said about the matter, I am sure that there was a certain feeling of tension throughout the ship that night. There is something about a torpedo striking a ship such as ours in the dark that seems to have a very permanent quality about it. We got through the night all right and the next morning at daybreak we could see smoke ahead of us on the horizon. This came from fires on Kwajalein island we learned later. By nine o'clock we had come up to the island and entered the lagoon by one of the passes. Our own fighting ships were outside this lagoon pouring fire into the island. Army troops had gone ashore there the day before. We anchored about four miles off the beach and soon small landing craft began to come alongside with wounded. These were brought aboard by stretchers up the main gangway and, as they passed into the ship, were screened by a team of three medical officers who directed them to the proper wards. By that afternoon it was realized that we were too far off the beach to give the best service, and that the wounded had a long trip in small boats before they could reach us. Therefore we moved in to within about a mile of the beach. From this vantage point we truly had a box seat for the fighting ashore. We took casualties all night and all the next day and by six that evening were loaded and underway for Pearl Harbor with over six hundred casualties aboard.

Under such conditions everything is not always ideal for surgery, and a case of mine will serve to illustrate what I am sure has happened to all doctors who have worked under such conditions. During the process of taking patients aboard at Kwajalein I happened to step out on the quarterdeck at the same time that an eye case was brought aboard. He proved to have a laceration of the cornea running directly across its widest diameter. His was a very recent injury and I wanted to repair it as quickly as possible but a checkup showed that every operating table in the ship was full at the time, and would be for some hours. The man was put on an ordinary wooden table in the master-at-arms shack and some instruments were obtained. With the aid of the chief master-at-arms holding an ordinary flashlight, and one of the laboratory technicians acting as scrub nurse, an iridectomy was done and the corneal laceration covered by a conjunctival flap in an entirely uneventful procedure.

During, and after, these beachhead operations there is relatively little or no rest until the ship is about three days on the way to a rear area. In these first few days there is too much to be done in the way of caring for the patients, many of whom are in critical condition, to worry about resting. One cannot help feeling that these wounded lads have given much more than we have and it is up to us to give them our best. And here I should like to pay a word of tribute to two groups, first, the wounded American fighting man of whom nothing that has been said in praise is too extravagant. No matter how great the pain, nor how grave his condition, he thinks of his wounded comrades first and, as one of our correspondents has so ably said, "the wounded don't cry." This is in decided con-

trast to the wounded Japanese, and I know them almost as well as I know the Americans for we took three hundred out of Saipan at one time. And, secondly, I should like to pay a word of tribute to my own shipmates for their dogged devotion to duty, their ability to work long hours, stretching into days, in operating room and wards, without rest and still keep going, whether it is operating or carrying food to the sick or being helpful in a hundred other different ways. I have watched lads of the black gang who have been standing a fire room watch for four hours, and I can assure you that is no fun in the tropics, come off watch and go up to the Officer of the Deck and volunteer for duty as a stretcher bearer, work for eight hours handling stretchers from small boats in heavy seas, and then go back to the fire room for their next regular watch only to repeat the process for days while we were loading. These, then, are the men with whom I have served, and they constitute the main reason why I shall always count it a privilege to have served in a hospital ship in time of war. I have seen them in health, and in sickness, and I have watched them die, and rarely is there one found wanting in any respect.

Cheap Glasses Cause Eye Distress.

Many poorly fitting and cheap glasses cause ocular distress and headaches, according to the December 7 issue of *The Journal of the American Medical Association.*

"A person who has such distress when he wears glasses naturally takes them off, just as a man takes off a pair of shoes that does not fit him comfortably," *The Journal* states in answer to a query.

In discussing the harmful or beneficial effects of the average sun glasses on the eyes, *The Journal* states:

"The average sun glasses, obtained in drug and ten cent stores, if sufficiently dark, are as a rule comforting to the eyes of the average wearer on an average bright day, on the water or on the beach, where they are usually used. They reduce the total amount of light entering the eyes. The idea that certain makes of glass do not transmit the 'harmful' ultraviolet light is mostly sales talk, as the ordinary white glass allows but little ultraviolet light to pass through. In fact, in order to obtain passage for ultraviolet through a window pane it has to be specially made for that purpose. A certain amount of ultraviolet light is beneficial to the eyes; it is for this reason that vacations are taken at the seashore and in the mountains, and in the winter patients are sent to sunny climes."

Arrest Tumor of Bone Marrow.

Two new English drugs, used in a New York hospital, have proved effective in checking multiple myeloma, a malignant tumor of the bone marrow.

No effective form of therapy was known for this highly fatal disease until these drugs—Stilbamidine and Pentamidine—demonstrated their tumor controlling and pain relieving properties.

Writing in the January 18 issue of *The Journal of the American Medical Association,* Isidore Snapper, M.D., from the Second Medical Service of the Mount Sinai Hospital, New York, reviews the treatment of 15 patients.

He states that "all these patients were suffering excruciating pains when the treatment was begun. Thirteen were immobilized in bed. All 15 improved considerably as far as the pain itself was concerned. Eleven could walk at the time of discharge from the hospital."

Dr. Snapper points out that the "treatment merely checks the disease and does not cure it."

This disease, usually associated with anemia, causes neuralgic pains. Later, painful swellings appear on the ribs and skull and spontaneous fractures may occur.

Injections into the veins of Stilbamidine proved successful in the majority of patients. However, in two who were not helped by Stilbamidine, Pentamidine was effective.

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MEDIASTINAL SYMPATHOBLASTOMA (NEUROBLASTOMA)

Report of Two Cases With Necropsies and Review of the Literature

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The mediastinum is an infrequent primary site of sympathoblastoma. Because only thirteen papers reporting fourteen well documented cases could be found in a review of the literature, it was thought advisable to add two of our own radiologically studied cases, both proven by necropsies.

Previous reports listing fully the age, sex, exact location, metastases when present, gross and microscopic appearances have been made by Anderson and Shennan,[1] Bridge,[2] Capaldi,[3] Cushing and Wolbach,[4] Frost and Wolpaw,[6] Hartung and Rubert[7] (2 cases), Lee and Ritter,[8] Sailer,[10] Scott and Palmer,[11] Stern and Newns,[12] Wahl and Robinson.[13] Wahlgren and Rudberg,[14] and Wissler.[15] Historically, in all discussions of mediastinal tumors of the sympathetic nervous system, the papers of Friedrich[5] and Loretz[9] are mentioned. However, both of these authors reported cases in which the predominating cells of the tumors were ganglioneuromas. Likewise, the recent excellent review of Sailer[10] recorded 13 cases of sympathetic tumors occurring within the thorax, only seven of which showed a preponderance of cells warranting the diagnosis of sympathoblastoma. Wahl and Robinson,[13] in 1943, in discussing ganglio neuroma and neuroblastoma state that, "neuroblastoma of the mediastinum is less frequent, but 13 cases have been reported out of approximately 200 cases of neuroblastoma in the entire literature." Two authors cite another paper as reporting four mediastinal tumors in a series of forty neuroblastomas, but a study of the original article showed that the mediastinal tumors were metastases from primary adrenal sites.

In the fourteen well documented cases found in the literature, there were 7 females, 4 males and in three, information as to sex was not available. The ages were 12 weeks, 5 and 22 months, 2, 2½, 4, 5, 5, 7, 38, 44 and 65 years, with one infant and one child the exact ages not known. One patient was colored. Our two cases were in white males aged 68 and 23 years.

The tumor sites were in the upper right posterior thorax near the lung apex in the lower cervical and upper dorsal region in five cases, the upper left posterior thorax, in lower cervical and upper dorsal region in two cases, the right posterior lower thorax in two cases, the left posterior lower thorax in one case, the superior central mediastinum in one case, the sixth dorsal laminae in one case, and in two the sites not known to us. Our two cases were somewhat centrally placed in the posterior superior mediastinum.

The gross size was not given in one case, not known in two cases, and descriptions varied from ovoid masses, 4.2 cms. in length and 2.8 cms. in diameter, to masses 13 by 13 by 7.5 cms. with frequent descriptions varying from orange size to pigeon's egg and bean size. Diameters of 5 or 6 cms. were most frequent.

Nine cases showed metastases and in most instances these were extensive. Three showed no metastases and the case reported by Cushing and Wolbach,[4] originally operated upon by McGuire in 1911 with microscopic finding of sympathoblastoma and reoperated upon by Cushing in 1921 with microscopic diagnosis of ganglioneuroma is known to be living in Richmond, Virginia, in 1946, thirty-five years after the original operation. Our case in the older man showed only thoracic metastases, but the case in the younger man showed thoracic and abdominal metastases.

Our first case showed only slight demonstrable changes in the width of his mediastinum following roentgen therapy with, however, moderate symptomatic response with diminution of venous engorgement and swelling of his neck. Our second case responded moderately well to deep roentgen therapy and lived seven years from the onset of symptoms and five years after the beginning of radiation treatment. During the last three years the mediastinal and thoracic tumors became increasingly resistant.

CASE REPORTS

Case 1.—H. E. F., a 65-year old white bricklayer, was seen in the Out-Patient Clinic in August, 1942, and the findings of "disorientation, cardiac enlargement, and auricular fibrillation" noted. He

failed to return to the clinic until June, 1944, six months after a fall which knocked him unconscious. During the six months after his injury, he had a total of five convulsive seizures each followed by a short period of amnesia. He denied any history of previous epileptiform seizures. There was no family history of epilepsy or other familial diseases.

On June 12, 1945, the patient was again seen in the clinic and was referred to the hospital because of painless swelling of the face and neck of two weeks' duration. His arms had begun swelling 2-3 days before admission. There had been no swelling of the lower extremities. He complained of mild dyspnea, but had had no cough, dysphagia, voice changes or weight loss. Admission temperature was 98° F., pulse 94, respiration 20, blood pressure 108 systolic and 80 diastolic. He had moderate pitting edema of the face, neck, and arms with slight edema of the upper thorax. There was marked distention of the neck veins and some venous distention and telangiectasis of the chest wall anteriorly and posteriorly. There was slight cardiac enlargement with distant heart sounds and with irregular rhythm caused by auricular premature beats.

Laboratory Data: The Kline test was negative; non-protein nitrogen 27 mgm. per 100 c.c.; hemoglobin 90 per cent, white blood count 4,500 with 52 per cent polymorphonuclears and 42 per cent lymphocytes. Urine examination was also negative. The venous pressure in the left arm was 422 mm. saline.

Chest film on June 11, 1945, and fluoroscopy had shown a moderate cardiac enlargement and slight, diffuse enlargement of the thoracic aorta. There was very slight widening of the upper mediastinum with no pulsation in this area and no displacement of trachea of esophagus.

On June 21st biopsy of a right axillary node was made. The pathological report was, "reticulum hyperplasia of lymph node."

Roentgen therapy was begun four days later and a total of 600 r (using 200 KVP, 20 ma., 50 cm. distance, thoreus No. 1 filter and a 20 x 20 cm. portal) was given to anterior and posterior mediastinum in 200 r (air) doses with one portal daily. Dyspnea and swelling improved slightly in five days and the patient was discharged to the Out-Patient Clinic.

Three months later, enlarged, bilateral anterior cervical nodes were felt and 200 r given to each side of the neck.

In January, 1946, he began having pain in the back of his neck and in both shoulders radiating down his arms. He had lost about 20 pounds in weight since his discharge from the hospital and had developed an exhausting cough only slightly productive of mucoid material. Some peripheral cyanosis was noted.

He became progressively worse with severe dyspnea, and he was readmitted to the hospital on February 2, 1946. The physical findings were those of massive left pleural effusion with remarkable distention of the veins of the neck and upper extremities and of the superficial thoracic and paraumbilical veins with generalized peripheral cyanosis. The venous pressure was more than 50 cm. saline in both arms. Routine blood and urine examinations were negative. Electrocardiogram showed auricular fibrillation and severe myocardial disease.

Thoracentesis was done on February 27th and 1,500 c.c. of clear amber fluid, specific gravity 1.015 was removed. A thick white clot formed in the fluid immediately after removal.

The patient died the following day.

Necropsy (only chest and abdomen permitted).

Exterior: There was very marked engorgement of all the superficial veins of the neck, arms, chest, and abdomen. A subcutaneuos node 1 cm. in diameter was found in the right chest wall below the clavicle in the mammary line.

Pericardial Cavity: The pericardial surfaces were smooth, moist, and glistening. The pericardial sac was filled with 400 c.c. of clear yellow fluid.

Pleural Cavities: About 1,400 c.c. of clear brown fluid was obtained from the left pleural cavity and there was a thin, yellowish membrane over the entire pleural surface on that side. On the right side, 880 c.c. of clear yellow fluid was obtained.

Mediastinum: In the superior central mediastinum was a firm, oval, nodular mass about 8 cm. in diameter and fixed to the surrounding structures. It was bounded laterally by the hila of both lungs and superiorly by the arch of the aorta. The mass extended inferiorly to 2 cm. above the auricles and posteriorly against the bifurcation of the trachea partially surrounding it laterally and extending up along the trachea for 3 cm. above the bifurcation. There was extension of the mass 3 cm. below the bifurcation of the trachea and along the right and

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left main bronchi anteriorly for 4 cm. on either side. There was no evidence of pressure and no gross involvement of the epithelial lining of the trachea or bronchi. The lymph nodes in this area were large and granular, some measuring as much as 3 cm. in diameter. Hilar nodes and the mass occluded by pressure the right middle lobe bronchus. The tumor mass enclosed and completely obstructed the superior vena cava 2 cm. above the right auricle and extended up along the wall of the vena cava for 3 cm. There was invasion of the lumen of the vessel with definite involvement of the intima. The mass within the lumen of the vessel had a papillary appearance at either end. The inferior vena cava was not involved. The pulmonary veins entering the left auricle were surrounded by the growth with no constriction of the left veins but with the right pulmonary veins constricted so that their opening into the auricle was slit-like, 1 cm. in width and 1 cm. in length. There was no apparent involvement of the vessel endothelium.

Lungs: The left lung was a dark, blue-brown color and was partially collapsed, feeling firmer than normal and appearing somewhat granular. The apex was adherent to the chest cavity posteriorly. On section, the lung substance was a dark brown-red color and very moist. Brownish, frothy fluid was expressed. The right lung was a pale red-gray color with patchy black areas. The upper lobe was partially collapsed and the middle lobe completely collapsed. There were several small emphysematous blebs about 2 cm. in diameter in the right apex. Infrequent, small, nodular masses were palpated in the upper and lower lobes and upon section; the masses were black and gray in color. There was a firm, meaty mass, scarred in appearance, approximately 2.5 x 3 cm. in the periphery on the lateral inferior border of the upper lobe.

Heart: There was definite right and left auricular and right ventricular dilatation. The coronary vessels were not remarkable. The left ventricle measured 1.5 cm. in thickness. The mitral valve had small, firm nodules about 2.5 mm. in diameter attached to its outer edge. The aortic valves were slightly thickened.

Spleen: The spleen was small, soft, and pale blue-gray in color with a wrinkled capsule. The pulp was a dark, reddish gray color and very granular.

The adrenals, stomach, intestines, pancreas, and gall-bladder were normal.

Liver: The liver weighed 1,600 gm. and was pale, brownish gray, finely granular, with liver edges sharp. There was a small black area 2 cm. in diameter and resembling a hemangioma on the left outer lobe.

The bladder and prostate were normal.

The abdominal aorta had a firm, gritty feeling with yellowish deposits under the endothelium. The renal arteries were tortuous and firm.

Microscopic Description: Sections of the superior vena cava showed the tumor mass extending into the wall of the blood vessel. The tumor cells were large and consisted of granular nuclei with no cytoplasm. There were several large areas of necrosis. The Foot silver stain showed no fibrillar processes and only a very few fibers. The Masson stain showed a very few fibrous cells within the tumor. Mallory's stain showed no glia. The tumor mass was seen in a lymph node with the immature neurogenic cells arranged in rosettes throughout. Many of the cells were somewhat club-shaped and elongated. Sections of the lung showed the alveoli filled with polymorphonuclear leucocytes, fibrin and a homogenous gray substance. The alveolar walls were edematous and injected. There were a few "heart failure cells" present. One area showed a fibrosed, homogenous area of inactive tuberculosis. All lung sections showed a mild degree of anthracosis. The skin node and bronchial and tracheal nodes all showed anaplastic cells similar to those in the tumor mass in the mediastinum. Section of the kidney showed patchy scarring with fibrosis of the glomeruli and tubercles in a V-shaped lesion the apex of which pointed into the kidney. All of the vessels were injected and the tufts were injected and full of red blood cells. The spleen showed an abundance of red blood cells with a decrease in the lymphoid cells and some increase in fibrous tissue. A section of the liver showed degeneration with moderate hemorrhage around the central vein. There was fibrosis in the aortic valve. The abdominal aorta showed a thick, homogenous gray area in the intima.

Pathological Diagnosis: Mediastinal neuroblastoma invading and totally obstructing the superior vena cava and partially obstructing the right pulmonary veins. Metastases extended to the tracheobronchial lymph nodes and to the skin over the right chest. There was marked distention of the collateral

venous circulation of head, trunk, and upper extremities. Hydrothorax was bilateral and there was hydropericardium. An old healed tuberculous lesion of the right upper lobe was noted at the lateral inferior border, as was emphysema of the lung at its right superior border. Benign nephrosclerosis was marked, and there was a mild generalized arteriosclerosis.

Case 2.—K. A., a 23-year old white male, was first admitted to the Medical College of Virginia Hospital on October 11, 1941, because of right pleuritic pain of three weeks' duration and low-grade fever. His illness apparently began, however, in April, 1938, when he had an illness characterized by a febrile course of several days' duration and thought to be influenza. Later that year, he again had some fever which was accompanied by right chest pain with radiation to his shoulder and back. Several days after the pain began, he was found in a state of collapse, and a diagnosis of "acute dilatation of the heart, probably produced by excessive physical effort," was made. He was hospitalized for approximately two months. After that, he did well until August, 1939, when he had what was thought to be malaria. In January, 1940, while he was working in Kentucky, he had another attack of what was thought to be pneumonia. Some straw-colored fluid was drawn from his chest at that time. In July, 1940, there was a recurrence of chest pain and fever. A chest film was made, and he was given x-ray treatments every 2-3 days to a total of approximately 12 treatments, with rather marked improvement clinically.

Examination upon admission revealed evidence of right pleural effusion and engorgement of the right jugular vein. Urine examination was negative. The Kline test was negative and there was no anemia. The white blood count was 14,420, with 64 per cent polymorphonuclears, 25 per cent lymphocytes, 8 per cent eosinophils and 3 per cent mononuclears.

Thoracentesis on the right was done on October 13th, and 120 c.c. of bloody fluid obtained. No tumor cells were seen in the fluid and smears were negative for tubercle bacilli. A chest film after thoracentesis showed a marked density extending out from the right hilum with oval densities in the right first interspace and at the left hilum—thought to be possible metastases. He was discharged October 21st, somewhat improved.

On July 17, 1942, he was again admitted to the hospital because of right chest pain, pleuritic in character, dyspnea, and low-grade fever. He had had moderate night sweats but no cough. Chest film upon this admission showed the density at the right hilum and lower cardiac border to be smaller than before but with two smaller nodes at the right hilum and a large oval nodule adjacent to the right dome of the diaphragm. These had the appearance of metastatic nodules of some new-growth. The heart and great vessels were not enlarged. Routine laboratory data was not remarkable.

Electrocardiogram was within normal limits.

Bronchoscopy was done on July 21st and there was no evidence of an intra-bronchial lesion. Both sides were thoroughly examined. There was a little bit of secretion coming from the posterior division of the right lower lobe bronchus, but there was no evidence of infiltration in the bronchial wall.

Roentgen therapy was given to the anterior and posterior chest, right and left sides, with a total of 1,000 r (air) to each of the posterior portals and 900 r (air) to each of the anterior portals, giving 100 r and 200 r doses to each of two portals daily, using 200 KV, Thoreus 1 filter, 20 ma., 50 cm. TSD, and 20 x 20 cm. portals. He improved fairly rapidly and did well for about seven months with a slight decrease in the size of the right hilum and lower right lung nodules. Then he began going slowly downhill. Roentgen therapy was repeated in March, August and November of 1943 and in January, 1944.

In spite of x-ray treatment, the pain in his right chest became frequent and nagging and repeated fluoroscopies showed no change in the right lung and hilum nodules. He occasionally expectorated bloody fluid and upon two occasions (October 20, 1943, and November 2, 1944) he expectorated tissue fragments which were diagnosed as "reticulum-cell sarcoma". After the third course of roentgen therapy, only occasional small doses of 100 r or sometimes 200 r were given over the right hilum and right lower lung nodule.

During the last six months of his life, he was almost completely bed-ridden with nagging right chest pain radiating to his back, severe dyspnea, cough, anorexia and pitting edema. He was again admitted to the hospital March 21, 1945, and died the following morning in acute respiratory distress.

Necropsy: (Only chest and abdomen permitted).

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Exterior: There was moderate emaciation with moderate pitting edema of both ankles, marked edema of the left arm and hand and less marked edema of the right upper extremity. There was a small healing ulcer about 1 cm. in diameter on the sacrum. The mid-thoracic spine curved markedly to the left with compensatory scoliosis in the lumbar region.

Thorax: The pericardium was about 5 mm. thick anteriorly and was adherent by fibrous adhesions to the epicardium. The left lung was atelectatic but there were no left pleural adhesions. The mediastinum was densely adherent to the midline of the sternum about 4 cm. inferior to the gladiomanubrial joint. There were manually inseparable adhesions to the right lateral, posterior, and diaphragmatic surfaces. Externally, the left lung was red and non-crepitant and the right lung was gray and firm. A firm, gray mass of tissue was palpated in the upper anterior mediastinal space and it extended cephalad to encase the innominate and the left subclavian veins. The mediastinal lymph nodes measured up to 3 cm. in diameter and were firm and gray.

Heart: The myocardium and valves appeared normal with no thickening of the left ventricular wall. A soft, gray, polypoid mass extended from the main right pulmonary vein into the right auricle and nearly filled the cavity of the auricle.

Lungs: The left lung was firm and red with a red, frothy fluid exuding from the cut surface. Only a small patch about 6 cm. in diameter in the upper lobe was crepitant and gray. The medial margin of the lower lobe was firm, dark red, and well-demarcated from the remainder of the lobe. On the right, the visceral pleura was firm, white to gray, and varied from 5 mm. to 1.0 cm. in thickness. It was adherent to the parietal pleura and was continuous with the inter- and intra-lobar septa. In the lower lobe posteriorly were a number of confluent areas composed of atypical degenerating and yellow zones forming a mass about 5 cm. in diameter which extended upward to the hilum. In the diaphragmatic pleura was a nodule 4 x 2 cm., very firm and apparently partly calcified. The posterior pleura of the lower lobe was firmly adherent to the adjacent ribs and intercostal tissue for an area about 6 cm. in diameter. The trachea and main bronchi were apparently normal but the neoplasm encroached upon the right lower lobe bronchus posteriorly.

Abdomen: The peritoneal cavity contained about 100 c.c. of straw-colored fluid. The liver was densely adherent to the medial surface of the right diaphragm in an area 2 cm. in diameter. The viscerae elsewhere were smooth, moist and glistening, and appeared normally placed.

The spleen, adrenals, stomach, pancreas, and gallbladder appeared normal.

Kidneys: The kidneys weighed 190 gm. each. There was a firm yellow depression measuring 2 cm. in its longest diameter in the upper pole of the right kidney. A similar, smaller area was found in the lower pole of the right kidney and one in the upper pole of the left kidney. Upon section, the depressed areas were about 6 mm. in thickness. The kidneys appeared otherwise normal.

Liver: The liver weighed 1,200 gm. The lower margins were sharp and glistening except for a diaphragmatic adhesion noted above. It was brownish-red and firm. Upon multiple section, the surfaces were firm, brownish-red and the periphery of the lobules deep red and well-defined. There were a few small areas of gray, firm tissue measuring up to 2 cm. in the upper and lower poles of the liver.

The bladder, prostate and intestines appeared normal.

Abdominal Aorta: The abdominal aorta was reduced in caliber to about 2 cm. by a cuirasse of firm, gray tissue. The intima showed a few small yellow plaques measuring about 2 mm. in diameter.

Microscopic Examination: Numerous sections through the tumor tissue in the mediastinum, heart, and lungs had the same appearance. The cells were large, pale, and contained rather elongated to round vesicular nuclei. The cells varied little in size and staining quality but showed numerous mitotic figures. They tended to arrange themselves in groups to form not only pseudo-rosettes but also true rosettes. The myocardial fibers were very edematous and fragmented. In one section, taken through the auricle, the lining of the epicardium was thickened by fibrosis apparently due to organization of an old thrombus. Between the areas of fibrosis, there were a few small fragments of typical tumor tissue. The lungs were marked by the presence of nodules of characteristic tumor tissue, some growing within the bronchi and apparently extending in this man-

ner into the lungs. They had grown in a concentric manner, compressing the surrounding pulmonary tissue, thus producing marked atelectasis. In some areas taken from the lungs, the pleura showed very dense fibrosis and hyalinization, apparently the result of repeated radiation, but within the alveoli were numerous foam cells and edema fluid. Aside from chronic passive congestion demonstrated by destruction of the central portion of the lobules by a marked congestion and the presence of a few polymorphonuclear leucocytes in these areas, the liver tissue showed replacement by the same type of tumor seen in the mediastinal neoplasm. Here again, concentric growth was demonstrable by pressure upon the surrounding tissue. Sections of the kidneys showed normal organs except for an area of ischemic infarction where the cells and nuclei had lost their differential staining powers and the tissue had assumed a pale, homogenous pink staining of coagulation necrosis. A section of the intestine showed complete autolysis of the lining mucosa. Bone marrow from a few sites showed a normal, active marrow.

Pathological Diagnosis: Mediastinal neuroblastoma involving the right cardiac auricle, the right lung, the pleura, diaphragm, liver, mediastinal and peri-aortic lymph nodes; accretio and concretio pericardii; pitting edema of the extremities; small decubitus ulcer of the sacrum; ischemic renal infarcts; scoliosis of the spine.

SUMMARY

Fourteen cases of mediastinal sympathoblastoma were found in a review of the literature and two more cases with necropsies have been added. Our two cases were considered to be lymphomas until the final microscopic studies were made. Sympathoblastoma should be constantly kept in mind in the differential diagnosis of all mediastinal tumors.

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XUM

PERSISTENTLY SERO-POSITIVE REACTION FOR SYPHILIS IN CHRONIC BRUCELLOSIS*

CONRAD L. GOSSELS, M.D.,
Mount Jackson, Virginia.

This report seems to me of general interest because it compels us to reconsider diagnostic procedures in a new light.

The patient is a 40-year old male. He always has been a nervous type. He had frequent spells of "nervous colitis". Several attacks of sore throat had led to a tonsillectomy. No other diseases or operations are recorded in his history.

In 1939, when his eyes were tested for glasses, a routine blood test showed up positive. When the patient denied any history of infection, this test was repeated and was negative.

1941, the patient had a general check-up that was completely negative except a positive blood test. When a control test was negative the physician advised the man to forget about it.

In summer, 1944, the patient consulted me for diarrhea that responded to routine treatment.

In September, 1944, cramps in his legs, difficulty of walking after leaving the bed in the morning, radiating pains into his legs and general weakness were noticed.

In October, 1944, the patient had fever in the evening. X-ray showed some peribronchial infiltration of the lower lobes that developed within a few days to an infiltration of the right lung. There was no response to penicillin. The fever dropped gradually without special therapy, and the lungs cleared up. The infiltration was considered a virus pneumonia. Blood Kahn and Wassermann tests done at that time were ++++, spinal puncture and Wassermann negative. Blood count: Hb. 91%, Eryth. 3640000, Leucoc. 9600, Band cells 5, segment. 66, lympho 24, mono 4, eosino. 1. Urine negative. Agglutination: negative. Normal recovery of the patient.

During 1945, the patient felt weak and got tired quick. No complaints, but some diarrhea occasionally. X-ray of G-I tract was normal. A general check-up, in December, 1945, showed a low blood pressure of 95/60 mm. Hg. Hemoglobin 78% (Sahli), Eryth. 4200000, Leuc. 9050. Normal differential count. Increased sedimentation rate (Westergren) 45 in the first hour. Kahn tests in two different laboratories showed one positive and one doubtful.

On January 20th, 1946, the patient was taken acutely sick with temperature of 102°, diarrhea, nausea, some vomiting and cramps in his abdomen. This acute gastro-intestinal infection responded to routine treatment.

On the second day of this sickness agglutination for typhoid (H-antigen) was positive 1 : 40 (rapid slide agglutination test, Health Dept., Luray), but there were no clinical signs of typhoid. On the 7th day agglutination for typhoid was negative (Health Dept., Richmond), but positive 1 : 20 for undulant fever. Stool, urine, and blood cultures were negative, but Kahn test was ++++ again.

The patient consented to going to Johns Hopkins Hospital, in Baltimore, for further study. He still denied any history of luetic infection and was worried very much about the different positive blood tests. Clinical studies were made at the department of Dr. J. E. Moore, Dr. C. F. Mohr and Dr. F. W. Reynolds, with these results:

Sedimentation rate 35 mm. per hour.

Hemoglobin 13.0 G.; hematocrit 42.1; WBC 5450; differential WBC juveniles 7; polymorphonuclears 53; eosinophiles 5; lymphoc. 31; monoc. 4; urinalysis completely normal.

Stool cultures were negative for enteric pathogens, and microscopic examinations of the feces were negative.

Sero-diagnostic tests were done and gave the following results:

Wassermann doubtful; Kahn positive; Eagle positive (titer 1 unit); Mazzini doubtful. Specimen sent to Dr. Neurath of Duke University showed the blood negative with Kahn, Kline, Mazzini and cardiolipin tests.

Spinal fluid was entirely negative. Heart was normal in size and shape by X-ray and fluoroscopy; circulation time was normal. Electrocardiogram showed no abnormalities.

Agglutination for B. abortis was positive in a di-

*Read before the Medical Society of Northern Virginia, at Front Royal, Va., August 13, 1946.

lution of 1 : 320. Brucella fixation skin test was positive.

Two months later, the serologic reactions showed Eagle doubtful; quantitative no unit; Kahn doubtful; Mazzini positive; Wassermann negative.

A recent re-check (October 1946) showed the conventional tests positive again, but with a new cardiolipin antigen the serologic test was entirely negative.

These findings led to the conclusion that here is one of the most persistently positive biologic false tests. It seems likely that the cause of the persistently positive serologic tests may be chronic brucellosis, although it is unusual for any syphilitic reagin to be present in the blood over a period of seven years. The markedly positive agglutination can best be explained by the fact that the acute illness of last January was an acute flare-up of chronic brucellosis, and added strongly to the agglutination titer in the blood-serum.

The clinical facts about brucellosis are known; very often no other symptoms except weakness can be found. Because of the chronic condition even the agglutination test did not prove helpful in former examinations. Only the acute flare-up led to the exact diagnosis.

The persistency of the biological false positive test was significant and led almost to a wrong conclusion. These false tests are known in more than 10% of cases in leprosy, malaria, infectious mononucleosis, smallpox vaccination, relapsing fever and atypical pneumonia. Recent reports showed a false test in 20 of 100 cases with simple nasopharyngitis. Fluctuations of the serologic titer in false positive reactors make the diagnosis still more difficult.

Although post-infectious positive reactions occasionally last as long as three months, most become negative within a few days or weeks. Transient false positive reactions may occur in apparently normal persons without recent illness, as found in many examinations for the Selective Service. Persistently positive reactions may occur in non-syphilitic patients over many years, but a length of 7 years as in this case is unusual. But there are not many so eminently chronic diseases as undulant fever is.

Many of these cases with positive reaction have been mistakenly diagnosed and treated as latent syphilis. The psychologic, social and legal consequence to the individual may be serious. While many false positive tests will be revealed as transient during a test-period of at last 3 months, there is no verification test available today to help in the diagnosis of those which remain positive.

For the practicing physician remains only one thing: repeat the serologic test at intervals and, if possible, use a quantitative flocculation test as the increase or decrease of the titer is of diagnostic importance. In the biologic false positive cases the titer will be fairly low, usually not exceeding 60 to 100 units, and will show a tendency to decrease in repeated tests, pointing towards a negative result. However, in the case of syphilis the titer may be higher than 100 units and tend to rise for a period of many months and remain at high levels of 200-300 units. In the biologic false positive cases this titer will never be higher than 5 to 50 units after several weeks.

Our old belief in the infallibility of the serologic tests has been shaken, and we have to adjust our knowledge to these recent developments.

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SIMPLIFIED RUBIN TEST TECHNIQUE

NORMAN G. PATTERSON, M.D., F.A.C.S.,
Bristol, Tennessee-Virginia.

There are two approved methods of performing Rubin's test for determining the patency of Fallopian tubes, and both of them have objectional features.

The method in which x-ray-opaque solution is used involves x-raying the patient, which is not convenient in many operating rooms. It also involves some danger to the patient and adds to the expense of the examination.

The second method, in which air or oxygen is used, is unsatisfactory to the beginner particularly, and rarely definite to the experienced unless air is found under the diaphragm by x-ray or assumed to be there by painful sensations. For a beginner particularly, it is very difficult to state positively that air bubbles heard with a stethoscope are coming through the Fallopian tubes and not leaking through the cervical canal.

The following technique has been used with complete satisfaction for the past five years, and seems fool-proof, and very simple. The apparatus must be air-tight. This is proven by occasionally submerging the apparatus in water and checking for leaks. The patient is either given anesthesia, or not, depending on the surgeon. It can be done either way. The tenaculum is applied and cannula inserted in the usual manner. The cervix is then depressed with the tenaculum and the vagina is filled above the level of the cervical os with a sterile solution, either water, saline, or antiseptic. Air is then pumped into the apparatus and manometer reading taken. If there is a fall in manometer reading, you know the air is going somewhere. If there is no leak in the apparatus and manometer reading falls, then the tubes must be patent since any leakage around the cervical os can be readily detected by bubbles escaping through the solution. Consequently, if no bubbles are seen as the manometer reading falls, then the tubes are patent.

Electric Power Building.

Headaches Relieved.

Sodium amytal, a sedative, will relieve such symptoms as headache resulting from tension or anxiety within one to five minutes when injected into the veins, according to three New York doctors writing in the current issue of the *Archives of Neurology and Psychiatry,* published by the American Medical Association.

The authors—Capt. Samuel Susselman and Capt. Fred Feldman, Medical Corps, Army of the United States, and S. Eugene Barrera, M.D., Albany, N. Y. —state that when symptoms are persistent and no organic cause can be discovered, the patient should receive an injection of sodium amytal. They claim that this sedative is of diagnostic value because it is a quick method of separating symptoms of organic disease from tension symptoms.

In more than 80 patients, treated over a period of nine months, the hospitalization period was shortened for many because of the use of this sedative.

"The patient in whom the test is clearly successful will have no residuum of distress but will express, often spontaneously, great relief and complete freedom from pain and a feeling of well-being," according to the physicians. They state that "this rapid lifting of discomfort is especially impressive to both patient and physician in cases of long-standing headache, which have remained unrelieved for months, or even years."

The authors point out that not only will this sedative relieve symptoms due solely to tension but it also will bring relief to patients with organic disease whose symptoms have been intensified by tension and anxiety.

THE FORUM

Is Partial Gastrectomy Desirable in the Treatment of Duodenal Ulcer?

TO THE EDITOR:

I read with great interest the guest editorial in the December 1946 issue of the VIRGINIA MEDICAL MONTHLY by Dr. Walter L. Palmer, Professor of Medicine, University of Chicago School of Medicine. Dr. Palmer is, of course, a gastro-enterologist of national standing and a man of wide experience in duodenal ulcer. A challenge of his opinions is somewhat bold.

I am not alone, however, in feeling that his conclusion, even though expressed as a prediction only, is a little too positive in the present state of our knowledge of vagotomy. As he himself points out, the operation of gastro-enterostomy for a good many years was considered "quite satisfactory", and now we know that it is not. The experience with vagotomy in any large group of cases is less than three years old. At the present time we are in the bandwagon stage of this operation, but even now cautious surgeons and internists are mounting the bandwagon with increasing reserve.

In considering vagotomy it must not be forgotten that we are depriving almost the entire gastro-intestinal tract of its parasympathetic supply. The ultimate result over years is not yet known; and it is quite conceivable that functional disturbances of moment may develop which are not yet foreseen. In our own small series of cases there have already been three instances of acute cardiospasm.

No surgeon is content with the results of any surgical procedure so far devised for the recalcitrant ulcer. As Dr. Palmer points out, however, the numerical disadvantage to ulcer patients as a whole is minor. Only about 10 percent of ulcer cases ever reach the surgeon. If the incidence of recurrent ulcer after partial gastrectomy is 8 per cent, that means that only .8 per cent of ulcer patients are really troublesome cases. The evidence concerning recurrence of ulcer after vagotomy is not yet complete. In our series there is one case of such recurrence. If in a large series after several years the recurrence rate is below the 8 per cent that Dr. Palmer quotes, then vagotomy obviously has an advantage in this important particular over partial gastrectomy.

In a recent discussion of the subject before the Southern Surgical Association, Dr. Frank H. Lahey asked for a conservative approach to this problem. He suggested further that the operation for the time being be limited entirely to cases without evidence of obstruction in order to disassociate the effects of vagotomy from the additional factor of a gastro-enterostomy. This seems to be a wise suggestion in our present stage of knowledge. On the other hand, Dr. Palmer speaks of vagotomy combined with posterior gastro-enterostomy as though it were an established procedure.

It appears to at least one surgeon that Dr. Palmer has rather cavalierly dismissed an operation, subtotal gastrectomy, which, although obviously not perfect, has brought permanent relief to an overwhelmingly large number of those subjected to it and has left as the ultimate duodenal ulcer problem only the most difficult of all cases. I would not challenge his statement that vagotomy may be the solution in this group, but I would suggest that too positive conclusions are not yet justified. It will be extremely unfortunate if vagotomy becomes soon so popularized that patients demand it even when surgical judgment is against it. The immediate relief of pain that occurs following vagotomy will make such popularization easy. Dr. Palmer's remarks are, of course, addressed only to the medical profession. They would, however, lead the casual medical reader to conclude that vagotomy is a definitely established procedure which has permanent value. This we certainly do not yet know.

EDWIN P. LEHMAN, M.D.

Charlottesville, Virginia
January 4, 1947.

TO THE EDITOR:

Thanks ever so much for your letter of January 9th enclosing Dr. Lehman's letter of comment on my guest editorial entitled: "Is Partial Gastrectomy Desirable in the Treatment of Duodenal Ulcer?"

Dr. Lehman's comments are of great interest. He is quite right in warning against the over-popularization of vagotomy. The ultimate result of the procedure over the years is not yet known. It is conceivable that untoward functional disturbances may develop. My own opinion, based upon the admittedly limited evidence to date, is that they will

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not be as frequent or as troublesome as those occurring after subtotal gastrectomy.

It will be recalled that vagotomy and posterior gastroenterostomy were advocated only as more desirable than subtotal gastrectomy for so-called "intractable" duodenal ulcer. Dr. Lehman very correctly deduces that I do not like subtotal gastrectomy. Perhaps this is because I have been unduly impressed by the difficulty in dealing with the recurrent ulcers and the occasional reflex or functional disturbances.

It must always be remembered that the great majority of patients with duodenal ulcer can be treated satisfactorily by medical measures alone. The frequency with which surgical treatment is indicated is, to a considerable extent, a matter of opinion and hence no reliable figures are available. My own opinion is that operation is desirable in less than 10 per cent of all patients with duodenal ulcer. In this group, vagotomy and posterior gastroenterostomy seem to me preferable. If jejunal ulcers develop, they are more amenable to medical measures than are those recurring after subtotal gastrectomy and, if necessary as a last resort, a subtotal gastrectomy could still be performed!

WALTER L. PALMER, M.D.
Chicago, Illinois
January 17, 1947.

Poison Honey.

We are inclined to believe that anyone who gets sick after eating honey has just had too big a bait, and we overlook the fact that honey may contain a serious poison.

A few years ago an early drought caused a shortage of honey producing plants to one old and experienced bee keeper. Mountain Laurel (generally called Mountain Ivy here) was about all his bees had to work on. This honey was declared good by a state gathering of bee men, despite the fact that the owner himself got sick on it before selling any. Later some half a dozen people became sericusly ill. The symptoms were the same in all. Fortunately vomiting was one early symptom or probably all would have died. There was a slow pulse and very weak at the wrist. One old gentleman of 75 had a pulse rate of 30 and heart sounds so weak they could hardly be heard and pulse so weak it could hardly be counted. He was found lying on the floor, unconscious and vomit all around him. The pulse rate of younger patients was about 35.

Laboratories could not confirm the poisonous natures of this honey due to lack of facilities, but it was generally conceded to be Laurel (or Ivy) poison. Stock that eat ivy during big snows get sick the same way and sometimes die.

Probably a dry Spring may be the time to watch closest for poison honey due to Laurel (Ivy).

A. B. GRUBB, M.D.,
Cripple Creek, Virginia.

Isolation of Polio Virus.

Successful isolation of what they believe to be nearly pure polio virus has been accomplished by two Stanford University scientists, Dr. Hubert S. Loring and Dr. C. E. Schwerdt. Laboratory tests, including photographs taken through an electron microscope, indicate that the isolated virus is 80 per cent pure or better.

The experiments, carried on in the Department of Chemistry laboratories at Stanford, were financed by the National Foundation for Infantile Paralysis, sponsors of the annual March of Dimes drive, from funds allocated to research on polio.

Production of purified virus is seen as an important step in the long and to date unsuccessful laboratory struggle to produce a vaccine effective against poliomyetitis.

Now that a virus which is relatively pure is available, the experimental door is open, Dr. Loring pointed out, to render the pure virus non-infectious by chemical or physical treatment, and, the scientists hope, to produce a concentrated vaccine free of the impurities which have plagued previous polio vaccine experimenters.

He cautioned against any conclusion that isolation of the pure virus will lead necessarily to production of a successful vaccine against polio, and stated that a long and exacting experimental path lies ahead of scientists seeking this vaccine.

According to the Stanford scientists, one of the contributions to the knowledge of polio which has resulted from this work is that scientists now know what the virus looks like, know how big it is, and what its chemical characteristics and properties are.

PUBLIC HEALTH

J. L. ROPER, M.D.,
State Health Commissioner of Virginia

The report of the Bureau of Communicable Diseases of the State Department of Health for December 1946, as compared with the same month in 1945, and for the period of January through December, 1946, compared with the same period in 1945, follows:

	Dec. 1946	Dec. 1945	Jan.-Dec. 1946	Jan.-Dec. 1945
Typhoid and Paratyphoid	7	8	123	170
Diarrhea and Dysentery	151	112	2,711	7,102
Measles	228	238	13,443	1,625
Scarlet Fever	193	456	2,724	4,246
Diphtheria	50	98	551	577
Poliomyelitis	11	6	142	335
Meningitis	12	10	204	215
Undulant Fever	9	2	70	32
Rocky Mountain Spotted Fever	0	1	92	99
Tularemia	20	9	77	50

WHOOPING COUGH

Examination of medical records and death certificates will readily show the frequent complications and number of deaths from whooping cough, the latter being found most concentrated in the under-two-year group. In many cases, while no open sign is present, the disease has affected body cells predisposing to later illness and disability.

The attention of the profession is therefore respectfully directed to the use of human and rabbit hyper immune anti-sera, available for prevention and therapeutic use. This biological is passive and temporary in action and is indicated for the non-immune with history of recent exposure, or for those who are in the early stages of whooping cough. Use beyond the second week is questionable, but may be tried if necessary with increased dosage. Usually one dose is sufficient for prevention unless exposure occurs ten days after injection. One or two doses will suffice for treatment except for advanced cases.

Satisfactory response to treatment should manifest itself in lessened numbers and intensity of paroxysms, and lower and shorter febrile reaction. Anti-pertussis serum is given intra-muscularly in concentrated form with dosage of about 3 c.c. Allergic reaction to the human serum is practically nonexistent, and rare with the rabbit serum. In the latter instance, rabbit sensitivity should be checked. Children who have been previously immunized should receive booster doses of concentrated pertussis vaccine. The serum may then be used if whooping cough develops.

At the present time the State Department of Health does not stock any make of anti-pertussis serum, but may do so in the near future. The biological may be secured through any of the following:

Children's Hospital Serum Exchange, Philadelphia, Pennsylvania
Human serum—unconcentrated—20 c.c. dose

Cutter Laboratories, Berkeley, California
Human serum—concentrated

Ayerst McKenna Harrison, New York City, N. Y.
Rabbit serum—concentrated

Wyeth Laboratories, Baltimore, Maryland
Rabbit serum—concentrated

New Wyeth Reference Booklet.

Wyeth Incorporated, Philadelphia, has just published "Therapeutic Agents of Special Merit", a handy new reference booklet for the busy doctor. "Therapeutic Agents of Special Merit" has been designed to provide the doctor with clear, concise and accurate information about selected Wyeth therapeutic agents, which by reason of their merit and widespread use, have proved to be helpful to the general practitioner as well as the specialist.

Each therapeutic product is illustrated and fully described, pointing out what it is, how used and how supplied. Copy has been held to a minimum. Only essential facts about each product are given. Additional clinical and laboratory data on the agents listed are available on request.

Included are convenient table of contents, and a comprehensive medical cross index.

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WOMAN'S AUXILIARY TO THE MEDICAL SOCIETY OF VIRGINIA

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Auxiliary News.

NORTHAMPTON-ACCOMAC

The first meeting of the New Year was held on January 14th at the home of Mrs. J. Walker Jackson at Machipongo. There were twenty-two members and one guest present. A delightful luncheon and social period preceded the business meeting at which the president, Mrs. B. N. Mears, Belle Haven, presided. Mrs. John R. Hamilton, Nassawadox, was appointed chairman of *Hygeia* for 1947. Mrs. J. L. DeCormis, Accomac, president-elect of the State Auxiliary, gave a report on the State Convention at Virginia Beach and the Auxiliary to the American Medical Association in Chicago. She then presented the silver vase attendance trophy to the Auxiliary. As they had won it for five times, it now becomes their property. It was voted that the vase remain with each president during the term of her office. Mrs. Rooker J. White, Keller, chairman on legislation, gave a timely and interesting talk on legislative matters, following which the meeting adjourned. The April meeting will be at the home of Mrs. G. L. Fosque, Onancock.

CATHERINE RUSH TROWER (MRS. HOLLAND),
Chairman, Press and Publicity.

RICHMOND

The annual Christmas party of the Richmond Auxiliary was held on December 13th. This was under the direction of the following members: Mrs. George Welchons, Mrs. Thomas Wheeldon, Dr. Matilda Chalkley, Mrs. Walter Rein, Mrs. A. F. Bagby, and Mrs. Benjamin Sheppard. Members brought toys which were donated to the Crippled Children's Hospital.

MYRTLE G. MORTON (MRS. W. R.),
Publicity Chairman.

Floral Eponym (47)

CLUSIA

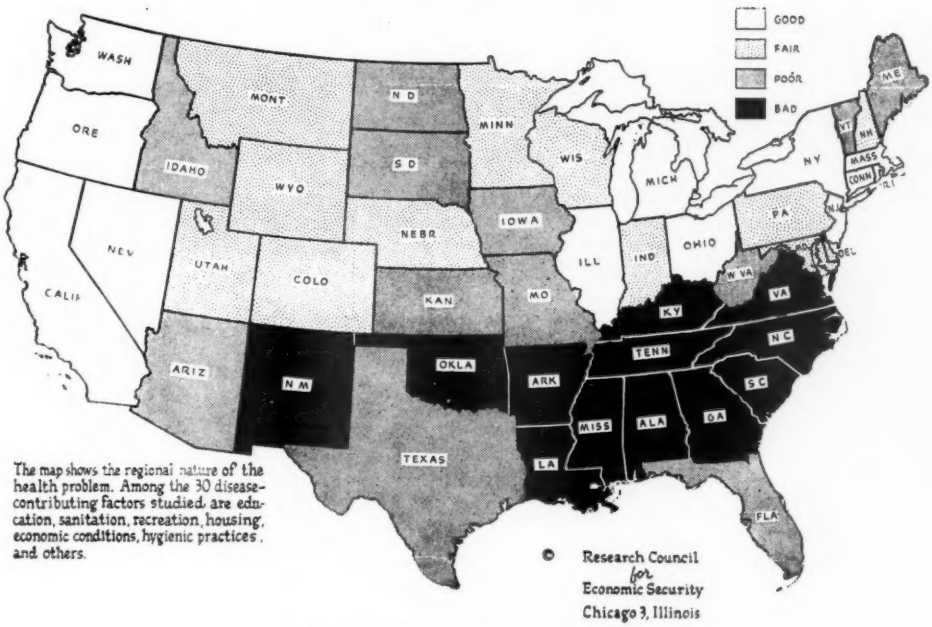
CHARLES DE L'ECLUSE, 1526-1609

CHARLES DE L'ECLUSE, or the Latinized Clusius, was a well-born Belgian who studied at Louvan, Marburg and Wittenberg. In the last he became acquainted with Melanchthon, the theologian. In 1850, Clusius came to Montpellier where his studies took a new direction under William Rondelet, a renowned physician and scientist. He graduated in medicine in 1553. He was physician to Maximilian II and Rudolph II and court botanist in Vienna. He broke many bones in his many botanical excursions and was a cripple when in 1593 he was called to his final post in Leyden. He was considered the greatest botanist of the 16th century.

Clusia is a genus of trees and shrubs found principally in Central and South America. Many are epiphytic climbers, the stems enveloping and ultimately smothering their hosts. Some, such as *C. rosea*, yield medicinal resins.

EDITORIAL

Our National Health Problem



The map shows the regional nature of the health problem. Among the 30 disease-contributing factors studied are education, sanitation, recreation, housing, economic conditions, hygienic practices, and others.

Infant Deaths in Virginia

INFANT mortality is considered one of the best criteria of efficient health service. It reflects the economic status and hygienic intelligence of the population, the character of the milk supply and to a lesser extent the water supply and the effectiveness of the control of infectious diseases as does no other single factor. The weakness of this measuring rod of public health efficiency is that its accuracy depends on the perfect recording of births, since the rate is based on the number of live births. Suppose, for instance, a community, with a rate of 59 per 1,000 live births, fails to record 10 per cent of its births (a not uncommon happening), the rate in that case would be 65.5 instead of the actual 59.

According to *Vital Statistics of the United States, Part II,* 1940 of the U. S. Census there were in the United States 47 deaths of infants under one year of age per 1,000 live births. At the same time Virginia had a rate of 59. Forty states had a lower rate than Virginia. The highest rate, 100, was in New Mexico and the lowest rate, 33, occurred in Oregon and in Minnesota. Virginia's rate is too high and her rank among the States is too low. Only seven States (Alabama, Arizona, Colorado, Louisiana, New Mexico, South Carolina and Texas) had a higher rate. One's first impulse is to blame the poor showing upon the negro population, but a glance at the figures shows that there is no close correlation between the infant deaths and the percentage of non-white population. Virginia with 24.7 per cent non-whites had an infant death rate of 59, whereas, Mississippi with 49.3 per cent non-whites had an infant mortality of only 54. Colorado with a non-white population of only 1.5 per cent had an infant mortality rate of 60 and New Mexico with 7.4 per cent non-whites had 100 infant deaths per 1,000 live births.

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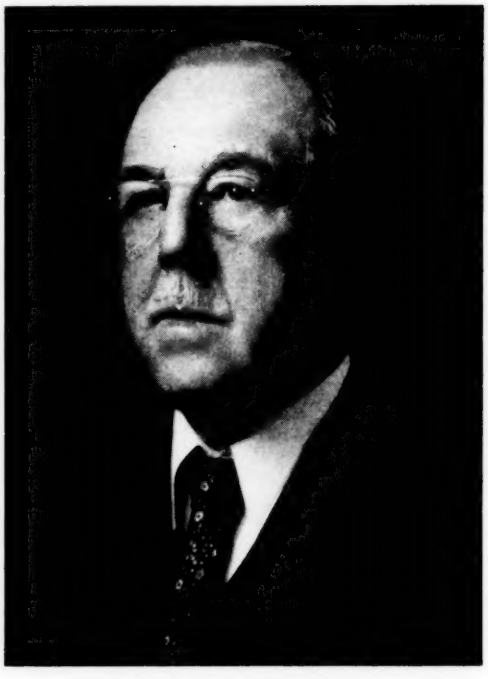
It is gratifying to record that in 1945 Virginia's rate fell to 48.5, but in the nation as a whole there was also a fall in the infant mortality. The rate for the United States for 1944, which is the latest available figure, was 39.8. It might be of interest in this connection to note that since 1936, when the first well-baby clinic was established in Virginia, public interest has grown until now there are 139 such clinics. In October 1942 arrangements were made to hospitalize babies needing hospital care, when such care was beyond the means of the family. During 1945-46 three hundred and two babies were hospitalized. The average stay in the hospital was 14 days and the average cost was $81.46.

John William Preston, M.D., 1867-1947

THE news of the unexpected death of Dr. John William Preston at his home on January first, came too late to put more than a notice in the January MONTHLY. Most doctors now practicing in Virginia remember with pleasure Dr. Preston's kindly welcome at the beginning of their professional life when they took the "Board". For more than thirty-three years he has worked untiringly to elevate, and to keep elevated, the professional standards in our State. Not only has his official work on the Medical



JOHN WILLIAM PRESTON

Examining Board of Virginia been invaluable, but his life and professional conduct have been a beautiful example of what a doctor ought to be.

Dr. Preston was born at Glade Hill in Franklin County, Virginia, December 13, 1867. He received his pre-medical training at Milligan College, Tennessee, and graduated in 1893 from the College of Physicians and Surgeons of Baltimore, which in 1915 merged with the University of Maryland School of Medicine to become the University of Maryland School of Medicine and College of Physicians and Surgeons. He studied further at the University of Pennsylvania, the University of London and

in Germany. He began practice in the Pocahontas coal fields at Keystone, West Virginia, but in 1908 he moved to Roanoke, Virginia, where his talents were soon recognized. In 1913 Dr. Preston was appointed to membership on the Medical Examining Board and in 1917 he became secretary of the board, a position he held until his death.

Dr. Preston was always active in legislative matters pertaining to the practice of medicine. For many years he was a member of the Legislative Committee of the Medical Society of Virginia, and when he was not on the Committee he cooperated actively with that important group. In 1927 at its Petersburg meeting, the Medical Society of Virginia elected him its president. It was in his term of office in 1928 that postgraduate instruction was begun—a project that eventuated into the Department of Clinical and Medical Education. This facility was not confined to the members of the Medical Society of Virginia, but was made available to all doctors regardless of color.

Dr. Preston was active in local affairs as well as in the State Society. He was a member of the board of directors of the Roanoke Hospital and served as chief of its staff in 1925. He was a past chairman of the Virginia section of the American College of Physicians, and a diplomate of the American Board of Internal Medicine. He was also a Mason and a member of the Shrine, and a deacon and elder in the Presbyterian Church. During World War I he served as a member of the Virginia Medical Advisory Committee.

Dr. Preston is survived by his wife and two daughters.

SOCIETIES

Albemarle County Medical Society.

At the meeting of this Society on January the 2nd, the following were elected as officers for the ensuing year: President, Dr. J. Edwin Wood, Jr.; vice-president, Dr. W. Roy Mason; and secretary-treasurer, Dr. C. Marshall Lee, Jr., all of Charlottesville.

Fredericksburg Medical Society.

At the December meeting of this Society, the following were elected officers for the ensuing year: President, Dr. L. A. Busch; vice-president, Dr. N. Talley Ballou; secretary-treasurer, Dr. James G. Willis. All are of Fredericksburg. Dr. D. W. Scott was elected to membership and Dr. C. W. Sale was reinstated after having been retired from the armed services.

Lynchburg Academy of Medicine.

The Academy met at the Lynchburg General Hospital on January 6, at which time new officers for 1947 were installed as follows: President, Dr. F. Read Hopkins; president-elect, Dr. Harold Riley; vice-president, Dr. Eugene S. Groseclose; and secretary-treasurer, Dr. Robert H. Cox. Two new trustees are Dr. Joseph Houck and Dr. Harvey C. Brownley. All officers are of Lynchburg. Dr. Frank F. Ellis and Dr. Lewis F. Somers of Lynchburg, and Dr. Ashby Coleman of Altavista were elected as active members and Dr. C. R. Titus of Bedford was elected to associate membership.

At this meeting, an interesting paper on Perforated Peptic Ulcer was presented by Dr. Joseph Houck.

Richmond Academy of Medicine.

The following officers were installed at the January 14th meeting of the Academy: President, Dr. Douglas G. Chapman; president-elect, Dr. E. T. Gatewood; vice-presidents, Dr. Charles L. Outland and Dr. H. J. Warthen, Jr.; and recording secretary, Dr. Henry St. George Tucker, Jr. Mrs. Mary N. Godbold continues as executive secretary-treasurer.

Roanoke Academy of Medicine.

At the regular meeting held on January 6th, the following program was presented: Leiomyosarcoma of the Stomach with Case Report by Dr. George S. Bourne with X-Ray Interpretation by Dr. Charles D. Smith; Infectious Polyneuritis by Drs. Edwin J. Palmer and R. Earl Glendy; and Importance of Early Diagnosis of Several Orthopedic Conditions by Dr. E. R. Troxler.

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Rockingham County Medical Society.

New officers for this Society, who assumed office the first of the year, are: President, Dr. Galen G. Craun of Harrisonburg, and secretary-treasurer, Dr. B. W. Nash of Timberville.

Warwick County Medical Society.

At the meeting of this Society on December the 19th, the following were elected officers for 1947: President, Dr. Emanuel Greenspon; vice-president, Dr. W. A. Mitchell; and secretary-treasurer, Dr. William H. Woodson. All are of Newport News.

Virginia Peninsula Academy of Medicine.

Dr. J. Grafton Love of the Mayo Clinic, Rochester, Minn., addressed the Academy at its meeting on January 20 at Newport News. His topic was Protruded Intervertebral Discs: Their Role in Intractable Pain and Paralysis—Diagnosis and Treatment. Dr. John M. Meredith of Richmond and Dr. James L. Thomson of Norfolk were present and each discussed Dr. Love's paper.

Eighty physicians registered attendance at this meeting. Dr. Robert H. Wright, Phoebus, is president of the Academy, and Dr. Chester D. Bradley of Newport News, secretary-treasurer.

NEWS

Are You a Fellow?

The American Medical Association is going to celebrate its centennial in Atlantic City, June 9-13, 1947. Elaborate plans are being made for this celebration.

Only Fellows and Invited Guests are eligible to attend. Membership in your state society is the primary qualification for Fellowship. Fellowship dues and subscription to *The Journal of the A.M.A.* are both included in one annual payment of $8.00, which is the cost of *The Journal* to subscribers who are not Fellows.

If you are not a Fellow and plan to attend the Atlantic City session, which will be a milestone in medical history, you can save yourself considerable time and confusion when registering, if you will *write now* to the American Medical Association, 535 North Dearborn Street, Chicago 10, and ask if you are eligible to become a Fellow.

Personnel Changes, State Department of Health.

Effective December 16, 1946, Amherst and Campbell Counties were joined to Charlotte County, creating the Amherst-Campbell-Charlotte Health District, with headquarters at Rustburg. The Health Officer is Dr. S. S. Shouse.

Effective December 16, 1946, Fauquier County was joined to Prince William and Stafford Counties, creating the Fauquier-Prince William-Stafford Health District, with headquarters at Manassas. The Health Officer is Dr. J. W. Still.

Effective January 1, 1947, Caroline County was joined to Hanover County, creating the Hanover-Caroline Health District, with headquarters at Ashland. Dr. J. D. Hamner, Jr., is the Health Officer.

Effective January 1, 1947, Dr. Linwood Farley was appointed Health Officer of the Augusta County Health Department, with headquarters in Staunton.

Appointments to State Board of Medical Examiners.

Governor Tuck has just announced the following appointments:

Dr. Kenneth D. Graves of Roanoke for the unexpired term of five years, ending June 30, 1950, to fill the vacancy caused by the death of Dr. J. W. Preston; and

Dr. Chalmers Gemmill of Charlottesville for the unexpired term of five years, ending June 30, 1947, to fill the vacancy caused by the death of Dr. Lewis Holladay.

Married.

Dr. James Motley Booker, Lottsburg, and Miss Marilyn Scott, Cape Charles, January 11th. He is a graduate in medicine of the University of Virginia, class of 1941.

Dr. Jacquelin Marshall Harrison, Richmond, and Miss Alice Ridgely Fisher, Baltimore, Maryland, January 4th. He is a graduate of the University of Virginia, Department of Medicine, class of 1946, and is at present serving an internship at the Union Memorial Hospital in Baltimore.

Dr. Claude M. Lee,

Who returned from the mission field to his home in Charlottesville during the war, has resumed his work in China for a period of three years, and is

now at St. Andrew's Hospital, at Wusih, Kiangsu Province.

"Courage and Devotion Beyond the Call of Duty."

Through the cooperation of Mead Johnson & Company $34,000 in War Bonds are being offered to physician-artists (both in civilian and in military service) for art works best illustrating the above title, as applied to physicians in war and in peace.

This contest is open to members of the American Physicians Art Association and will be judged June 9-13, 1947 at the Atlantic City Session of the American Medical Association. For full details, write Dr. F. H. Redewill, Secretary, Flood Building, San Francisco, Cal., or Mead Johnson & Co., Evansville 21, Indiana.

Spring Graduate Course.

The Gill Memorial Eye, Ear and Throat Hospital, Roanoke, will hold its twentieth annual Spring Graduate Course, April 7th to 12th. All lectures will be held at the Patrick Henry Hotel, and operations and clinical demonstrations will be given at the hospital. The guest faculty includes Drs. A. J. Ballantyne, Glasgow, Scotland; Arthur J. Bedell, Albany; William P. Beetham, Boston; Eugene M. Blake, New Haven; E. B. Burchell, New York; Russell N. DeJong, Ann Arbor; Murdock Equen, Atlanta; William T. Hunt, Philadelphia; Chevalier L. Jackson, Philadelphia; Chester S. Keefer, Boston; Raymond E. Meek, New York; R. Townley Paton, New York; Lyman G. Richards, Boston; John R. Richardson, Boston; Bernard Samuels, New York; John M. Sheldon, Ann Arbor; C. R. Straatsma, New York; and O. E. Van Alyea, Chicago. Resident members are Drs. E. G. Gill; Gardner H. Landers; Robert M. Ferrell; Charles H. Dow; and James A. Campbell.

Dr. J. Arthur Gallant

Has returned from service in the U. S. Navy and resumed private practice in Richmond, with offices at 203 West Franklin Street.

News from the University of Virginia.

On January 6th, Dr. Fletcher D. Woodward, Professor of Otolaryngology, read a paper, prepared by Dr. Woodward and Dr. Thomas Holt, on the Use of Soluble Gauze (Oxidized Cellulose) in Otolaryngology, before the Southern Section of the American L. R. and O. Society, Miami, Florida.

Dr. Woodward made two talks on Infections and Tumors of the Maxillary Sinus before the Winter Postgraduate Course in Otolaryngology and Ophthalmology, Graduate School of Medicine, University of Florida, Miami, on January 7th and 8th.

West Virginia State Medical Association.

Due to the uncertainty concerning the date of the reopening of The Greenbrier at White Sulphur Springs, the Kanawha Medical Society has invited the West Virginia State Medical Association to hold the 80th Annual Meeting in Charleston. The Council has accepted the invitation and the meeting will be held in this city, Monday, Tuesday and Wednesday, May 12-13-14, 1947.

Error in Ciba-Medical Slyd-Rul.

Ciba Pharmaceutical Products, Inc., Summit, New Jersey, recently mailed to all physicians a handy slide rule for metric conversion. Since sending this out it has been found to contain an error in the computation—placement of decimal point of the conversion from 0.4 grain to gram. This should read 0.025 gram, not 0.25. A corrected paper table has been mailed which can be used temporarily.

Ciba regrets this error which causes doctors inconvenience and will replace the temporary slide with a new celluloid table as soon as possible.

Dr. B. B. Bagby, Jr.,

Formerly at Pine Camp Hospital, Richmond, is now Chief of Tuberculosis Service at the Newton D. Baker VA Hospital, Martinsburg, West Virginia.

News from Duke University, School of Medicine.

A month's course in Medical Mycology, under the direction of Dr. Norman F. Conant, is to be offered at Duke University School of Medicine and Duke Hospital, Durham, N. C., during July 1947. The course will be offered every day in the week, except Sunday, and has been designed to insure a working knowledge of the human pathogenic fungi within the time allotted.

Emphasis will be placed on the practical aspects of the laboratory as an aid in helping establish a diagnosis of fungus infection. Insofar as possible and as patients become available, methods of collecting materials in the clinic for study and culture will be stressed. Work with patients, clinical material, cultures and laboratory animals will serve as

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a basis for this course. Also, an opportunity to study pathologic material, gross and microscopic, will be given those whose previous training would allow them to obtain the greatest benefit from a study of such material.

The number of applicants for the course will be limited and the applications will be considered in the order in which they are received. An attempt will be made, however, to select students on the basis of their previous training and their stated need for this type of work.

A fee of $50.00 will be charged for this course, upon the completion of which a suitable certificate will be awarded. Please direct inquiries to Dr. Norman F. Conant, Duke University School of Medicine, Durham, N. C.

Dr. Eugene A. Stead assumed his duties as Professor of Medicine at the Duke University School of Medicine on January 1, 1947, filling the professorship held by Dr. Frederic M. Hanes whose untimely death occurred last March.

As an experiment in graduate teaching, the Department of Anatomy is offering a review of anatomy on a graduate level using motion pictures, lantern slides, and other visual aids. This review is offered as a part of the speciality program in Neuropsychiatry and Orthopedics. The course is conducted as round table discussions with panels including staff clinicians especially interested in the region of anatomy under discussion.

Appointed Schering Director.

Mr. Francis C. Brown, President of Schering Corporation, Bloomfield, N. J., has announced the appointment earlier this year of Mr. Basil O'Connor as a new member of the Schering Board of Directors. Mr. O'Connor is Chairman of the American National Red Cross and President of the National Foundation for Infantile Paralysis.

Announcement of Mr. O'Connor's selection was made to executives, office and plant employees at the Schering Annual Christmas Party in the Grand Ballroom of the Hotel Pennsylvania, New York, December 21, 1946. An additional feature of the event was the presentation to Schering personnel of an annual bonus totaling more than $135,000, largest in the company's history and in keeping with Schering's policy of dividing profits with employees.

The new Schering director is well-known for his national public health and welfare activities. Mr. O'Connor was the former partner of the late President in the law firm of Roosevelt and O'Connor, and is now senior partner of O'Connor and Farber, of New York. In addition to chairmanship of the American National Red Cross and presidency of the Georgia Warm Springs Foundation, he is President of the National Foundation for Infantile Paralysis, and participates in a number of civic, legal and educational activities.

VA Mental Hygiene Clinic.

Plans for establishing a mental hygiene clinic in Richmond during the spring were announced in January by the Veterans Administration. Out-patient treatment will be available to veterans with service-connected psychiatric or nervous disorders.

Dr. C. T. Wilfong, who has been acting as manager of the VA'S Richmond Sub-regional office, will head the clinic. He also will be chief medical officer for the sub-regional office.

The clinic, VA officials said, will be in operation about the middle of February. Out-patient treatment will be available here for veterans who feel some psychiatric or nervous disorder. Officials feel that such treatment will effect an early cure in many cases and will thus obviate the necessity for hospitalization.

Treatment will be provided for veterans on leave or discharged from VA neuropsychiatric hospitals, if they have service-connected disability.

Dr. Wilfong's switch from manager of the sub-regional office to chief of the mental hygiene clinic and chief medical officer is in line with VA's policy of utilizing medical personnel for medical duties. Heretofore much of his time has been consumed with administrative duties of a non-medical nature.

A diplomate of the American Board of Psychiatry and Neurology, Dr. Wilfong has a long record of service with the Veterans Administration. He is a graduate of the medical college of the University of Louisville, and he served as a medical officer in World War I. He is a member of the American Medical Association, of the American College of Physicians, and of the American Psychiatric Association.

China's Determination.

Political unrest in China has not affected the determination of men and women in all walks of life in that country, particularly in the medical pro-

fession, to speed the task of reconstruction, according to Dr. Hu Shih, former Chinese ambassador to the United States and now president of Peking University.

The report of Dr. Hu, who is honorary president of the American Bureau for Medical Aid to China, one of the participating agencies of United Service to China, states that American aid for Chinese educational institutions has a "stimulating influence of immeasurable value."

"There remains in China today," Dr. Hu says, "the same strong spirit of patriotism, self-sacrifice and devotion to duty which carried us through the difficult war years. This is especially true among our professional classes, our teachers, doctors, welfare workers and public servants.

"A similar spirit is also prevalent among our medical students. A large number of them are preparing, not for private practice, but for public service. Their ambition is to see in their own generation the development of China as a medically self-sufficient nation.

"I am glad that ABMAC hopes, in 1947, to increase the number of fellowships for promising young men and women pledged to become medical teachers. Travel grants and the exchange professorship plan also fit into the picture. It is imperative that our older teachers, after long isolation of war years, have the opportunity to visit foreign schools and hospitals to catch up on techniques developed in the past ten years. The ABMAC plan to concentrate future aid on five or six medical schools also fits into the hopes of our medical leaders to see China possessed of medical schools worthy to be ranked with the best of other countries."

Death Among Doctors.

Death claimed approximately 3,558 physicians during 1946 as compared with 4,015 the previous year, according to the January 11 issue of *The Journal of the American Medical Association.*

On the basis of an analysis of 3,358 obituaries published in *The Journal* during the year, it was found that the average age at death was 66.1 as compared with 65.3 in 1945. The majority of the physicians—584—died between the ages of 70 and 74, the same age group that was dominant in 1945.

Heart disease remains the leading cause of death among physicians. Coronary thrombosis and occlusion accounted for 738 deaths (655 in 1945), 283 occurring between the ages 60 and 69. Cerebral hemorrhage, thrombosis and embolism accounted for 404 deaths; cancer and tumors were responsible for 352 deaths and pneumonia for 199. Tuberculosis caused 31 deaths, asthma 10, bronchitis and pleurisy five, and other diseases of the respiratory system 31.

The Journal finds significant the total of 55 suicides as compared with the 25 recorded for 1945. There were 130 accidental deaths, 55 of which involved automobiles.

The 3,358 *Journal* obituaries included 40 physicians who were killed in action during World War II and 128 who died while in military service. A number of those who were killed in action had first been reported missing in action. Eight physicians lost their lives in the Asiatic area, seven in the Philippine Islands, six in the China Sea, five in Germany, five in the Pacific area, two in Italy, two in France and one each in Formosa, Luzon, North African area, Okinawa and Tinian. Nine were reported as prisoners of war.

Since the outbreak of World War II on December 7, 1941 *The Journal* has recorded the deaths of 257 physicians who were killed in action and 501 who died while in military service. Reports still being received by *The Journal* indicate that the aggregate total of 758 military deaths among physicians will be greatly augmented when the complete statistics are made available through official military channels.

For Sale.

GE X-Ray, Model 1775, 30 ma, 85 KVP, table with Bucky, in excellent working condition. James P. King, M. D., Radford, Virginia. (*Adv.*)

Medical Writing Service.

By experienced medical writer. Bibliographies, case histories copied, articles abstracted, editing, ghost writing. Reasonable rates for expert service. Mrs. F. A. Cooksley, Box 634, Franklin Station, Washington 4, D. C. (*Adv.*)

Wanted.

A McCaskey cabinet of the type sold to doctors about twenty-five years ago. Write "McCaskey" care the MONTHLY, 1200 East Clay Street, Richmond 19, Virginia. (*Adv.*)

Wanted—

A young doctor for a junior partner. Country practice; good salary plus commission. Address "Doctor", care this journal, 1200 East Clay Street, Richmond 19. (*Adv.*)

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OBITUARIES

Dr. Joseph Albert Abercrombie,

Prominent physician of Richlands, died January 11th following a cerebral hemorrhage. He was forty years of age and a graduate of Johns Hopkins University in 1930. Dr. Abercrombie was on the staff of Johns Hopkins Hospital for seven years and for two years during that time was assistant to the chief of neurosurgery. For the past ten years he had been connected with the Mattie Williams Hospital in Richlands where he specialized in brain and neurosurgery. Dr. Abercrombie was a member of the State Board of Health. He had been a member of the Medical Society of Virginia for ten years. His wife and two daughters survive him.

Dr. Thomas Benjamin Pearman,

Well known Richmond physician, died January 12th of heart disease, after an illness of two weeks. He was forty-nine years of age and graduated from the Medical College of Virginia in 1921. Dr. Pearman served as Richmond city physician from 1922 to 1934. He was considered an outstanding diagnostician and was a member of the staff of Grace Hospital. He was a Scottish Rite Mason and a member of Acca Temple and the Legion of Honor. Dr. Pearman had been a member of the Medical Society of Virginia for twenty-two years. His wife and two sons survive him.

Dr. John William Preston.

At the regular meeting of the Roanoke Academy of Medicine, on January 6, 1947, the following resolutions were adopted.

It is with regret that the Roanoke Academy of Medicine has learned of the death of Dr. J. W. Preston, which took place at his home here on the morning of January 1, 1947.

We wish to extend to his family our deepest sympathy. Not only will he be a great loss to his family, he will be a great loss to the Medical profession as a whole, to the State Medical Society and to this Society, where he filled many positions, from the president down, always to the very best of his ability. Kind, patient and thorough in all his undertakings.

It is hard to find words to express the love and esteem in which he was held by people in all walks of life who knew him.

Be it resolved that a copy of these resolutions be sent to his family, to the local paper and to the State Medical Monthly.

W. L. POWELL
W. R. WHITMAN
ALVAH STONE
Committee

Resolutions on Dr. Plunkett.

The Lynchburg Academy of Medicine wishes to acknowledge the loss of one of its oldest and most active members, Dr. Flavius Ogburn Plunkett, whose untimely death occurred December 18, 1946. In so doing the following resolution is offered:

BE IT RESOLVED that The Lynchburg Academy of Medicine wishes to express to the family of Doctor Plunkett their sincere sorrow in his untimely passing.

That the Medical Profession, particularly the specialty of Obstetrics and Gynecology, has suffered a tragic loss. From the beginning of his specialization, in which he was considered a pioneer in this community, he bent his every effort both mental and physical to the improvement of maternal and infant care, not only in this community but in the State and nation. In this capacity he served for many years as a member of the Maternal and Infant Welfare committee of the Medical Society of Virginia. His sincerity and devotion in his field of practice was unquestioned, and the welfare of the patient was never secondary to his personal feeling or gain.

The community has lost a very valuable and civic-minded citizen, whose interests were broad and who devoted a large part of his time to the welfare of indigent patients in the City Clinic and hospitals. He was largely responsible for the development and growth of the City and County Clinics for the needy.

BE IT RESOLVED that these resolutions be spread upon the minutes of this meeting, that a copy be sent to his family, and to the Medical Society of Virginia.

JAMES R. GORMAN, *Chairman*
EUGENE S. GROSECLOSE
SAMUEL E. OGLESBY
Committee

January 6, 1947.